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THE
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AMERICAN OTOLOGICAL SOCIETY.*

PRESIDENTIAL ADDRESS.

WESLEY C. BOWERS, M.D.,

New York.

Two years have passed since you honored me with the Presidency of the oldest national Otological Society in the world — an honor which I appreciate more than any words of mine can express. Ordinarily you would have had the opportunity of conferring the office on someone else at the Seventy-sixth Annual Meeting which was to have been held in June, 1943. Circumstances — for the second time since the inception of the Society in 1868—forced cancellation of a meeting. In 1878, a railroad strike kept members from attending the Tenth Annual Meeting at Niagara Falls. In 1943, the emergency of all-out war intervened.

In December, 1942, the terrific pressure on the railroads for the transport of men and materials of war led the government to request that no meetings not directly connected with the war effort be held. It was, therefore, decided at the Council meeting, held that same month, that the 1943 Annual Meeting be cancelled. During 1943 the power of the Allied Nations began to change the world situation unmistakably for the better. In December of that year the Council, after due consideration of existing conditions, felt that it was right and proper that we hold a meeting in June, 1944.

*Presented at the Seventy-seventh Annual Meeting of the American Otological Society, New York, June 5, 1944.

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The past two years have wrought tremendous changes in geography, politics, modes of work and living, and in our personal lives. Our approach to our profession is no exception. We have arrived at new or altered concepts of the treatment of many diseases, increased knowledge of preventive medicine and care of the injured. We shall be increasingly confronted with rehabilitation problems. It was deemed important, therefore, that we meet to co-ordinate and disseminate some of the vast amount of new-found knowledge accelerated by the war. We realized that travel conditions are still far from good and that only those members living within easy radius of New York might be able to attend.

One of the few bright spots in the world picture today is in the field of medical science. Constructive advances of great importance have been numerous in the past two years. Outstanding are the development and perfection of the blood bank; the use of blood substitutes, especially blood plasma, to counteract shock; and the discoverey of the Rh factor which has helped markedly in the avoidance of untoward reactions to transfusions.

The development of chemotherapy will undoubtedly be marked in history as one of the great medical advances of our century. It is generally believed that it takes 10 years to prove the ultimate value of any new drug or medical procedure. The urgent, concentrated drive of war emergency undoubtedly shortens this period. Although we have been using sulfa drugs for the past eight years, the past two years have greatly increased our knowledge of them. Many conditions, once considered hopeless, yield readily to sulfa therapy. It is a pity that such a potent remedy should be jeopardized by injudicious use. Many commercial firms are now advertising various combinations of sulfa drugs with vasoconstrictors, for use as a spray or drops for the nose and throat. When used indiscriminately and for relatively trivial purposes, there is always the possibility that the patient may become sensitized to the drug, and thus be denied the wonderful aid of sulfa when really needed for a serious dyscrasia, such as pneumonia, mastoiditis or meningitis.

Within the past four or five years, penicillin has become better known, with increasingly favorable reports on its

effect on infection. Limited production has, up to now, largely restricted penicillin to military uses, and much valuable data obtained by Army doctors has perforce remained a military secret. The average civilian physician has had very limited experience with penicillin. New plants and chemical techniques of manufacture (speeded, it may be noted, by military necessity) are now coming into operation and we may shortly anticipate penicillin for civilian use. Keefer feels that penicillin "is a very effective, relatively nontoxic, anti-infective agent for Gram-positive infections which are resistant to the sulfa drugs. It is also effective in pneumococcal infections and in gonorrhea." It is of less than no value in the great bulk of the Gram-negative infections, but we may reasonably anticipate that as other antibodies are developed the usefulness of this class of chemotherapeutic agent will be greatly extended.

Atabrin, replacing the practically unobtainable quinine, has not only saved many lives but has actually made possible our offensive in the South Pacific by mitigating the threat of malaria. When it is possible to release data accumulated by the Army Medical Corps it will be interesting to note whether atabrin is less deleterious to the ears than is quinine.

One result of advances such as the use of plasma and chemotherapy is that many more of the wounded will be saved in this war than in any previous war. It is not enough to bring them back alive; we must do all in our power to restore the disabled to normal lives. The field of plastic surgery — always so vital in wartime — is being vastly expanded. Otologists, long accustomed to delicate work in small, deep cavities under difficult conditions, should be well qualified, with little additional training, to do plastic procedures on face and neck.

Fistulization of the labyrinth, for correction of obstructive deafness, has been considerably improved in the last two years. Although this operation is not new, the intra-aural route has been in use for only six years. The problem of insuring permanence of the fistula is not quite solved, yet the fact that it is possible to maintain a fistula at all, and that this artificial opening does improve hearing, opens up a fascinating field for further study. While it is still too early to

draw definite conclusions, it would seem certain that some form of fistulization will hold a prominent place in our armamentarium.

All forms of acoustic trauma are vastly increased by the war. Marked advances in the prevention and treatment of deafness consequent to injuries of the hearing apparatus are to be expected when experienced gained by Army doctors is released. This is also true of the comparatively new field of aviation medicine, which is destined to increase in importance in the years ahead as aerial transport at high altitudes and the increased use of personal planes becomes commonplace to civilians.

With the advent of chemotherapy, the number of cases of mastoiditis and its sequelae — particularly meningitis — has steadily declined. Indeed, it would seem that meningitis is well on the way to becoming a rare and seldom fatal illness. Strangely, such a desirable advance has its undesirable aspect. The great decline in cases has already made it difficult to teach our resident physicians how to perform adequate mastoidectomies, sinus thrombosis and labyrinth operations, explorations of the petrous bone and brain abscesses. Since there will always be a few such cases, despite chemotherapy or other measures, all otologists will have to be capable of dealing with them. It may prove necessary to develop the required technique entirely on the cadaver.

Since it is becoming increasingly evident that allergy is the underlying cause of many misunderstood and puzzling conditions, and that allergic reactions are more frequently seen in the ears, nose and throat than in other parts of the body, it seems to be essential that all otologists should be thoroughly trained in allergic manifestations and treatment. This implies a fuller knowledge of general medicine, which in turn means better otologists.

The present military need for doctors in the armed forces has greatly curtailed training in the special fields of medicine. Indeed, it may be a matter of years before men can again be trained for these specialties as they were before the war. New supplementary methods of visual education may well come into vogue, as, for example, full-color television

screens upon which close details of a complex operation can be followed simultaneously by students in many medical schools. When peace comes, we of the medical profession shall truly, as Kaiser has said, "be starting from ashes to build a new world. Truly a great opportunity to make the world better than ever."

The program at today's meeting was planned with the idea of bringing us as much information as possible on the above problems. In these days of stress it is especially difficult to find the time and effort to prepare papers, and I know you all join with me in expressing a very deep appreciation of the willing and distinguished response of the contributors to today's program.

To our most efficient Secretary, Dr. Friesner, who has so ably handled the proceedings of the Society, I extend my heartfelt thanks for his invaluable aid during the past two years. I also thank the Council for the great help it has given us, and the membership for its unfailing cooperation and support throughout my term of office.

In closing, may I leave with you the words of Bernard Baruch: "This is truly a period of great changes, and coming events hold much in store for us. The period will be filled with greater opportunities than ever before. Let us apply our skills and our initiative in a united effort to protect, preserve and advance. With vision, courage, resourcefulness and energy, we will go farther than we have ever gone before."

17 East 61st Street.

THE OTOLITHS AND THE PART THEY PLAY IN MAN.*

W. J. McNally, M.D.,
Montreal.

The otolithic organs occurred much earlier in the scale of development than did the semicircular canals but the canals have attained a more important clinical rôle than have the otoliths. The canals are probably not any more important to the body economy than are the otoliths. A likely explanation is that the canals are more accessible to experimental operative interference than is the utricle, the most important vestibular otolith. Breuer, who first ascribed division of function to the parts of the labyrinth, regretted that he was unable to carry out uncomplicated removal or stimulation of the otoliths. This was at a time when he and many others had carried out important experiments upon the semicircular canals. Tests for semicircular canal function were developed very soon after the nature of labyrinthine function was understood, whereas there is not even yet a satisfactory test for otolithic function.

Structure of the Otolithic Organs: An otolithic end-organ consists of the otocyst or macula, which is the main supporting structure. It is usually saucer-shaped and covered with neuroepithelium—the sensive end-organ. On top of the neuroepithelium is the true otolith (ear-stone). This overlying structure varies considerably in its makeup. It may be a grain of sand as it is in the prawn. In the lower fishes it is a stone-like structure, whence comes the name. The otolith of the frog's saccular macula resembles a small bag of milk of magnesia. In most mammals and in man the otolith consists of an overlying membrane of greater specific gravity than the macula. It is unfortunate that the word otolith is used interchangeably to refer to the whole end-organ on the one hand, or to the true otolith on the other. Usually one can deduce from the text how the word is being used.

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*Read at the Forty-ninth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., New York, June 9, 1944.

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Otolithic Function: Breuer concluded from his experiments and deductions that the otoliths perceive linear movement and the position of the head in space. He referred to the otoliths as static sense organs. The early experimenters treated the otoliths as a group; there was little attempt made to distinguish between the various otolithic organs.

Importance of the Otolith: Kreidl's classic experiment which consisted of substituting iron filings for the ordinary sand otoliths in the otocyst of the prawn showed that movement of the iron otoliths by a magnet caused marked disturbances of the animal's equilibrium and suggested that it is the movement of the otolith on the hair cells of the otocyst which acts as a stimulus to the end-organ.

Bethe, Clarke and Prentiss concluded from experiments on some forms of crustaceans that the otocyst can function without an otolith, but they noted that in otolith-possessing animals the absence of the otoliths causes some disturbance of equilibrium.

In some of the lower animals there are as many as five separate otolithic organs. In the higher mammals, including man, there are only two otoliths: the utricular otolith which lies in the horizontal plane, and the saccular otolith which lies in a vertical plane.

Associated Extralabyrinthine Reflexes: Magnus and de Kleyn were responsible for distinguishing between labyrinthine reflexes on the one hand and extralabyrinthine reflexes on the other. They described neck reflexes and body reflexes which are closely allied to labyrinthine reflexes. From topographical studies and from experiments in which they centrifuged away the otoliths from the maculae in the guinea pig, they allocated certain reflexes to the saccule and certain reflexes to the utricle.

SACCULAR FUNCTION.

A survey of the experimental work done upon the saccule indicates that the saccule does not have an equilibrial function. Among the experimenters who came to this conclusion are Parker, Van Heusen, Maxwell, von Frisch, Stetter, Werner, Ulrich, Versteegh, Laudenbach, Brown, Tait, McNally and Huddleston. Additional experiments by Ashcroft and

Hallpike and by Ross and McNally in which they recorded action currents from the frog's saccular nerve, indicated that the saccule does not respond to vestibular stimulation but that it does respond to vibration stimuli. Because the weight of experimental evidence would indicate that the saccule is not concerned with equilibrium, any further remarks about the part that the otoliths play in many will have particular reference to the utricular otolith.

UTRICULAR FUNCTION.

The utricular otolith in man lies in the horizontal plane in very close association with the ampullae of the vertical and horizontal semicircular canals. The function of the utricle comes under the two different aspects suggested by Tait: *1.* Its propriation — how does it act? *2.* Its servation — what does it do?

There is still need of additional experimental work along both these lines but information as to how the utricle acts is particularly meager and scanty. There has been considerable controversy in the literature as to whether the utricle is stimulated by the pressure of the otolithic membrane, by the pull of the otolithic membrane, or by a displacing or torsion effect of the otolithic membrane. There is little experimental proof to support any of the various theories advanced and for that reason propriation will not be discussed further.

Utricular Reflexes: Magnus analyzed positional reflexes and, partly on the basis of topographical studies of the labyrinth, divided them into those arising from the utricle and those arising from the saccule. Magnus classified positional reflexes into: *1.* attitudinal reflexes — tonic labyrinthine reflexes on the body musculature, on the limbs and on the neck; *2.* compensatory eye positions, both vertical and rotatory; *3.* labyrinthine righting reflexes — asymmetrical and symmetrical.

Magnus used the term attitudinal reflexes to describe the reflexes which occur when the body passes from one attitude to another by a change in the distribution of tone in its static muscles. These attitudinal reflexes are not entirely labyrinthine in that they arise from a co-operation of the tonic labyrinthine reflexes with the neck reflexes.

Versteegh's experiments indicated that all the positional labyrinthine reflexes arise from the utricle.

Utricular Servation: A limited number of experiments have been reported in which an attempt has been made to stimulate or eliminate the utricle, and only those most free from experimental error are mentioned here.

Direct Stimulation of the Utricle: Maxwell exposed the utricles of the dogfish, leopard sharks and rays. He placed a small ball of cotton wool over the otolith of the utricle and then grasped the ball of cotton wool with a forcep and thus attempted to move the otolith on the macula. If he moved the cotton wool forward the eyes moved forward; this was contrary to his expectations based on the results of his tilting experiments. If the fish is tilted forward the eyes normally are elevated. From the results of his experiment he derived some ideas about the mode of action of the utricle but we will not follow his line of argument here. He concluded from all his experiments that there is no sharp differentiation of function between the otoliths and semicircular canals; he believed that they reinforce each other.

Ulrich operated upon the live pike; his experiments were very carefully controlled. He found that movement of the utricular otolith forward or to its own side elicited eye reactions which are in keeping with the reactions to tipping tests. Pressure on the utricular otolith elicited the ordinary compensatory eye movements which result from tipping an animal forward or to its own side. This is just opposite to Maxwell's results. Ulrich observed that moving the otolith to the midline did not cause any reaction. He reported that moving the otolith backwards was not free from technical difficulties. His experiments were most carefully carried out and his results were more in line with what one would expect from tilting experiments, etc. It is important to note that he reported that there was no nystagmus following uncomplicated utricular stimulation. Ulrich's experiments confirmed the findings of Tait and McNally that a utricle shows very little or no response when tipped to the opposite side.

Utricular Nerve Section: Versteegh succeeded in sectioning the nerve to the utricle on one side in a rabbit. All the reflexes ascribed by Magnus and de Kleyn to both the saccule and the utricle disappeared.

Tait and McNally developed a technique for severing the nerves to the frog's utricle on one or both sides without injury to other parts of the labyrinth. They could also sever the nerves to the semicircular canals and other otoliths, leaving only the utricles intact. From these experiments they concluded that the utricle gives origin to labyrinthine righting reflexes and tonic reflexes in the neck, body and limb musculature. The utricle does not respond to angular acceleration about a vertical axis, but it does respond to centrifugal force and is responsible for the lean towards the center of rotation. The utricle also responds to linear acceleration, as will be shown later. Each utricle in the frog is stimulated by slow tipping about the horizontal axis, forward and backward and to its own side. It is stimulated to a lesser degree by tipping to the opposite side. When stimulated by a slow tilt the utricle elicits a compensatory reaction of the body and limb musculature which tends to keep the head level. These two experimenters also found that the utricles in a frog are stimulated by a quick tilt about a horizontal axis but in the absence of all six semicircular canals the response is anticompensatory. It is in the wrong direction to protect the animal and is in the opposite direction to the reactions which normally follow semicircular canal stimulation.

A frog with only two utricles intact is disturbed by a severe swaying or pendulation on attempting any movement. This would suggest that, whatever the mode of stimulation of the utricles, they have an unsteadying effect when not controlled by the semicircular canals.

Action Currents from Labyrinth: Ross, who studied the electrical reactions from the utricular nerve of the frog, was able to confirm the presence of all the reflexes mentioned above except the anticompensatory reaction of the utricle to a quick tilt.

Utricular Response to Angular Acceleration: Tait and McNally attempted to explain or analyze the utricular anticompensatory reactions to a quick tilt. They had previously noted that an unopposed semicircular canal reaction is markedly exaggerated and greatly in excess of the normal protective reactions. If the utricle is removed from the frog on

one side only, the reactions from the vertical canals of the opposite side are hyperactive. This would indicate that the utricle of one side controls and damps down the reactions of the opposite vertical canals. Tait and McNally also reported that when a frog with only one single utricle remaining was subjected to a quick tilt to either side, the anticompensatory response which it gave was most marked when the frog was tilted to the side of the absent utricle. In other words, when the utricle is slowly tilted to its own side it gives a maximum compensatory response and when quickly tilted to the opposite side it gives a maximum anticompensatory response.

Centrifugal Force: When considering turning movements about a vertical axis one must remember that if the animal is any distance from the center or axis of rotation, centrifugal force develops, and Tait and McNally showed that the utricle is stimulated and does respond to centrifugal force.

Van Wulfften Palthe and Quix considered that centrifugal force stimulated the otoliths and that it is a factor in flying.

LABYRINTHINE TONE.

Ewald was the first to describe labyrinthine tonic influences and he pointed out that there are two distinct types acting upon the muscles. He reported that a pigeon after a bilateral labyrinthectomy showed two types of tonic disability. If its eyes were covered with a hood, *1.* it lacked a capacity for acting quickly; *2.* its head tended to be displaced when subjected to a steady pull or pressure. The displacement of the head was followed by changes in limb posture. Tait pointed out that this change in limb posture was brought about by the neck reflexes described by Magnus and de Kleyn. The loss of quick action has been shown by Tait and McNally to be in large part due to absence of the semicircular canal reflexes.

The stretch reflexes of muscle and the tendon reflexes, described by Liddell and Sherrington, are, in themselves, unable to protect an animal without labyrinths in a sudden emergency, such as landing after a jump. They are only local reflexes. It requires the rapid message from the semicircular canals to the whole body musculature to prepare the animal for a sudden muscular emergency.

Semicircular Canal Postural Tone: Tait and McNally have analyzed the postural changes which follow unilateral labyrinthectomy. There is an asymmetrical forced lean to the side which has been operated upon, for which the removal of the utricle is in large part responsible. It has been conclusively demonstrated, however, that removal of any individual semicircular canal imposes a characteristic residual pose on the animal. It is not a forced posture in that the animal is free to assume any other pose whatsoever, but the characteristic residual pose, attributed to the absence of the particular canal, is the more frequent. This is additional proof that the semicircular canals, as well as the utricles, are concerned in the maintenance of postural tone.

It has been shown elsewhere that both the semicircular canals and the utricular otoliths are concerned in the body's response to any sudden emergency. The canals and the otoliths check each other in order to maintain that degree of muscular tone necessary for normal posture and normal reaction to any sudden stimulus.

Utricles Not Constantly Signaling: Tait and McNally were able to show that the utricular maculae emit nerve impulses during head movements only and that the maculae remain quiescent so long as the head is stationary. If a frog is slowly tilted through an increasingly greater and greater angle, it makes, at intervals, new compensatory movements with its body musculature by which on each occasion the head is brought once more to or nearly to the horizontal. All the separate stationary postures involved can be artificially imposed upon and then held by a completely delabyrinthized frog. There is no need, therefore, to assume that the utricular maculae exert a continuous tonic influence, or in other words, are constantly engaged in signaling. They transmit their nervous signals only at particular junctures; namely, when the head is moved. During any head movement the semicircular canals and the utricles dictate the necessary muscular readjustment to return the head and eyes to their original level position in space and the new position is maintained by a holding mechanism of the muscles. The labyrinth, therefore, serves to dictate new positions necessary to compensate for movement, but it does not maintain these positions during rest.

NYSTAGMUS.

Positional Nystagmus: If the otoliths are not signaling when the head is at rest, any other part of the labyrinth might be equally responsible for a positional nystagmus. The relationship of nystagmus to the utricle has elicited a considerable amount of discussion. A very common complaint of a patient with equilibrial disturbance is that he can lie with the head in only one certain position, usually on one side, otherwise he becomes dizzy. It is sometimes difficult to ascertain from the patient whether it is the side position that is disturbing or whether it is the movement to that side that distresses. It is usually assumed that it is the position that upsets and utricular disease is thought to be the cause.

Writers have varied considerably in their opinions. Fischer and Wolfson have discussed the subject of positional nystagmus at length and they pointed out that Bárány, Voss and Ruttin assumed that this nystagmus which results from any special position of the head is due to a disturbance of the otolithic apparatus. Breuer, Stein and Grahe and some other authors believed that disturbance of the central nervous system or neck reflexes could be responsible for this type of nystagmus. In considering this matter it is important to note that there are no experiments on record where uncomplicated stimulation of the utricle elicits nystagmus. There are no necropsy reports showing utricular disease in patients who suffered from positional nystagmus or dizziness.

Labyrinthectomy and Positional Nystagmus: de Kleyn and Versteegh carried out experiments to analyze the positional nystagmus which follows alcohol poisoning in the rabbit. They found that the positional nystagmus disappeared after a bilateral labyrinthectomy and that it was affected by unilateral labyrinthectomy. It was not influenced by removal of the saccular maculae on one or both sides. They concluded that the origin of this nystagmus in the peripheral labyrinth must in some way be connected with the labyrinthine tonic mechanism, but they add that one must not, under any circumstances, conclude from this that normal nystagmus in the mammal arises from the otolithic organs.

Utricular Stimulation and Nystagmus: Ulrich did not notice any nystagmus when he stimulated the utricle in the

pike. Versteegh did not notice any nystagmus when he severed the nerve to the utricle in the rabbit.

Nystagmus and Motion Sickness: Bárány, Politzer and Neumann each stated that they had not observed nystagmus in man during seasickness. Wojatschek also remarked upon the absence of nystagmus in seasickness. Fleish reported some nystagmus when he subjected rabbits to horizontal linear acceleration. Sjöberg, though he suspected the presence of nystagmus in motion sickness, was unable with the naked eye to detect nystagmus in any form of motion sickness. Hasegawa, Magnus, de Kleyn, Ulrich and Versteegh have concluded from their experiments that stimulation or injury of the utricles does not elicit nystagmus.

The absence of nystagmus in motion sickness is one of the reasons why the otolithic mechanism of the labyrinth rather than the semicircular canals is regarded as the origin of the symptoms of motion sickness.

Deafmutes and Motion Sickness: James, Reynolds, Minor and Sjöberg all expressed the opinion that from their observations seasickness does not occur in deafmutes with nonfunctioning labyrinths. If such observations could be confirmed in a large number of well tested cases it would be strong proof that it is labyrinthine stimulation which is the main cause of motion sickness.

LINEAR ACCELERATION.

Magnus believed that straight line movements, linear acceleration, or as he referred to them — progressive reactions, were perceived for the most part by the semicircular canals. He stated that his experiments did not exclude the possibility that the otoliths might also be stimulated by linear acceleration.

The Utricle and Linear Acceleration: Breuer, Mach, Ewald, Lee, Ach, Wojatschek, Bárány, Fleisch, McNally, Tait, Groebbels and Hasegawa have concluded from their experiments and observations that it is the otoliths that are stimulated by linear acceleration. Groebbels and Tait and McNally have reported that after elimination of the semicircular canals in the pigeon and in the frog, normal reactions can still be elicited in response to linear acceleration. Tait and McNally and Ross were able to demonstrate that a frog which retained

only its two utricular maculae — all other parts of the labyrinth having been removed — is able to react strongly to linear acceleration.

Motion Sickness and Linear Accelerations: Bárány, Sjöberg and Wojatschek concluded from their observations and experiments that vertical progressive (linear) acceleration is the most disturbing factor in the production of motion sickness and that this movement stimulates the otoliths.

Motion sickness has been forced to the attention of the military authorities because of the increase of mechanization during the present war. It was decided that the available literature upon the subject should be reviewed. The review revealed very few scientific observations upon such a common ailment as seasickness.

ANALYSES OF SHIPS' MOVEMENTS.

Wojatschek (1909) stated that the complicated movements of a ship consist essentially of two basic patterns —an up and down linear movement and a side to side or fore and aft rolling movement. He concluded that the rolling movements involving angular acceleration and stimulating the semicircular canals are of relatively little importance in seasickness because they tend to annul each other when occuring in such rapid succession, a complete cycle in every two to eight seconds. He referred to an experiment of Bessemer in which a chamber was suspended in a ship to eliminate the effect of the side to side roll and yet an observer within became seasick. He concluded that it is the up and down movements of a ship, the progressive linear movements, in a vertical direction, which constitute the main stimulation bringing about seasickness and that these movements stimulate the otoliths more than the semicircular canals. These conclusions were derived from estimation of forces acting upon ships of different sizes compared with estimated thresholds of semicircular canal and otolithic stimulation. They were not supplemented by ablation experiments on the labyrinth of animals.

Quix (1922) calculated the forces acting on various ships. He assumed that angular acceleration stimulated the semicircular canals and that vertical linear acceleration stimulated the otoliths. Quix concluded that during the roll of the ship the stimulation is never sufficient to exceed greatly the

threshold values of stimulation of the semicircular canals. On the other hand, during up and down movement and during pitching, the threshold value for linear acceleration is greatly exceeded. The otoliths are thereby stimulated and the symptoms of seasickness are elicited. He explained the absence of nystagmus in seasickness on the grounds that stimulation is subliminal for the semicircular canals. Quix calculated that the value of g* varies greatly in different parts of a boat and he believed that this variability of otolithic stimulation is a very disturbing factor. He made the comparison that a flickering light, a tickle or an interrupted sound are more disturbing than a steady light, a steady touch or a constant sound.

Sjöberg (1929-1936) also made some mathematical calculations of the forces acting on different ships. He concluded that rotary acceleration in a ship is always below the threshold of stimulation for the semicircular canals. He subjected human beings and dogs to up and down movements in an elevator and was able to produce motion sickness. He noticed that deafmutes with dead labyrinths were immune to elevator sickness. He was able to make dogs motion-sick by up and down movements in a crane. After the removal of both labyrinths the dogs no longer became motion-sick. He concluded that linear movements stimulate both the otoliths and the semicircular canals and that both the canals and otoliths are factors in causing seasickness.

This is the main part of the evidence which indicated that vertical linear acceleration is the stimulus which affects the otolithic part of the labyrinth and causes motion sickness.

INVESTIGATION OF MOTION SICKNESS.

Early in the war the National Research Councils of Britain, Canada and the United States undertook the investigation of motion sickness with the co-operation of the navy, army and air services in all three countries. The late Sir Frederick Banting was one of the prime movers. There were two main objects in view: First, the selection of personnel — it was hoped that it might be possible to detect those abnormally susceptible to motion sickness; second, and most important, it was hoped to find a remedy for motion sickness.

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*The symbol g refers to the acceleration of gravity, 32 feet (976 cm.) per second.

Some work was authorized in an attempt to derive information about the mechanism of motion sickness. As the program progressed, this question of the mechanism of motion sickness became increasingly important because of difficulty experienced in comparing experiments between the different laboratories.

Apparatus: It was imperative at the outset to evolve some method whereby motion sickness could be produced in the laboratory in order that its effects could be studied. A simple swing received general adoption. When the various workers met to compare results, it was obvious that there was need for standardization both of equipment and of methods.

The simple two-point suspension swing may vary in the length of the suspending ropes, in the width of the arc, in the rapidity of the swing. It may be pushed by hand or uniformly motor-driven. The amount of the stimulus varies depending upon the position taken up by the subject on the swing, whether he is sitting up, lying down, whether his head is in the center of the swing or off center, whether the head is fixed or free to move, etc. Cipriani stated that a four-point suspension swing introduces tangential accelerations which are not present on a two-point suspension swing. Experiments on a high speed elevator, on a crane, on an up and down spring arrangement or on a see-saw all introduce different forces which must be calculated and assessed. Though the simple swing was most universally used, additional apparatus was devised to simulate other phases of ship and aeroplane movements.

Standardization: Andre Cipriani constructed an accelerometer which was used to measure the main forces acting on some of the swings. He pointed out that on a simple swing the acceleration, acting radially with respect to the arc of the swing, is the principal stimulus responsible for the production of swing sickness. He stated that there are in addition three components of tangential acceleration which he did not consider of sufficient magnitude to act as a labyrinthine stimuli. Cipriani calculated the radial acceleration as about 0.7 g at the point of maximum displacement when the length of the swing is 13 feet, the total angular displacement is 80 degrees (vertical displacement 3.3 feet) and the frequency is 15 complete cycles per minute.

Head Fixation: At the outset of the National Research Councils' investigations, McNally and Cipriani, from theoretical considerations, stressed the importance of fixing the subject's head during all motion sickness experiments. This was in order that the direction of the forces acting should have as nearly as possible a constant relationship to the planes of the labyrinthine end-organs. Abels, in 1926, noted that the disturbing effects of seasickness are lessened when the subject is lying down with his eyes closed. The effect of changing head position on the incidence of motion sickness or swing sickness was strikingly brought out during the recent investigations by J. G. Howlett. He was able to show that a susceptible subject is most likely to become ill on the swing when he is sitting or lying with the head erect. He is least liable to be made ill when the head is in such a position that a line passing through the ear canal and the external angle of the eye is in the vertical plane. These findings were confirmed by the experiments of G. W. Manning, G. E. Hall and W. G. Stewart.

D. McEachern and G. Morton reported that vertical movement in a high speed elevator produced motion sickness when the subject sat with the head erect. Lying down on the floor of the elevator with eyes directed to the ceiling reduced the incidence of motion sickness.

Similarity of Results: For practical purposes there was a large measure of agreement in the findings of the workers from the different laboratories, even though calculations showed slight differences in the forces acting on the various swings in use. The similarity was such as to allow some conclusions to be drawn about the efficacy of the drugs under investigation.

Variation in Results: Some very wide variations in results were also reported from different laboratories, particularly when apparatus other than the simple swing was used. Four different groups of experimenters found that when special machines were constructed to produce an up and down movement of an equivalent g to that calculated for the simple swing, they were unable to produce motion sickness in susceptible individuals. Other workers again reported having conducted experiments in a high speed elevator in which they

were able to produce the symptoms of motion sickness. Such variable results would indicate that an effective stimulus does not entirely depend upon the amount of *g*. Other factors must have some influence.

Complexity of Stimulation: It has been suggested that an interrupted stimulus is more effective than a continuous stimulus; that the relationship of negative to positive acceleration is important. The time of action of the acceleration probably has an influence: von Wulfften Palthe stated, with reference to angular acceleration, that the threshold of stimulation depends not only on the amount of acceleration but on the time during which the acceleration is acting. It may also be that the apparently unimportant tangential accelerations on the swing have an influence when coupled with a radial acceleration.

Coriolis Phenomena: An acceleration acting by itself may be ineffective, whereas when acting in conjunction with some other forces it may produce symptoms. Wojatschek (1935) described this as the Coriolis phenomena. In his experiments the patient was sitting on a turntable with the head and trunk bent forwards. Rotation about a vertical axis did not produce any pronounced symptoms; however, if during or just at the end of the turning the subject straightened up, bringing the head into the erect or vertical position, a definite lean to the side of the rotation immediately took place. This movement about a horizontal axis in conjunction with the turning about a vertical axis brought on a more pronounced reaction than a simple turn of the same magnitude about either of these axes singly. Wojatschek described this phenomena in his recent book (1943) under the heading, "Test with Double Rotation." If the reaction is very pronounced it is taken as an indication of a hyperactive labyrinth. Wojatschek considered that the otoliths play a part in this reaction but his evidence is not convincing.

All the above suggestions about the complexity of stimulation in motion sickness require accurate experimental analyses.

Measurement of the Forces Acting: There is a need to estimate with specially constructed accelerometers the forces

acting on a ship at sea, and on an airplane in the air. Various sizes of vessels should be studied under varying conditions of sea and air. If possible the measurements in each position should be associated with data collected from observing the incidence of motion sickness in personnel at these various points on the boat or in the airplane. It may be possible with such data to make accurate laboratory analyses of the component forces and to determine just what are the essential single forces or combinations of forces which go to produce motion sickness.

Attempts have been made to correlate the incidence of swing sickness with air sickness on the one hand and with seasickness on the other hand. These efforts have failed probably because of the many unknown factors which have just been discussed.

Habituation: Habituation to labyrinthine stimulation is not well understood. The subject was reviewed in full by McNally and Stuart in their review of the literature.

Desnoes, Dodge, Dohlman and Mowrer suggested that immunity to seasickness or habituation involves the central nervous system and may be connected with the learning process.

Some attempts have been made to reduce susceptibility to air sickness by giving preliminary swing treatment. The results have not been entirely satisfactory. The Russians claim good results in reducing the incidence of motion sickness by preliminary vestibular training.

Extralabyrinthine Factors in Motion Sickness: Some of the experiments of the National Research Councils' groups were designed to assess the importance of extralabyrinthine factors in the production of motion sickness. As the result of these experiments the theories that movements of heavy viscera, or blood pressure, or cerebrospinal fluid pressure changes might be responsible for motion sickness have been eliminated. It is generally admitted that visual, olfactory, gastronomic and psychological factors do play an important part in the production of motion sickness; but they are aggravating factors, not the basic cause.

Labyrinthectomy and Motion Sickness: W. J. McNally, E. A. Stuart and G. Morton found that four dogs, each of

which had previously vomited on several occasions while in the simple pendulum swing, were no longer susceptible after complete bilateral labyrinthectomy. These four dogs after operation were subjected to vertical movements in a high speed elevator together with one normal dog. The four labyrinthectomized dogs were completely unaffected by the elevator motion, whereas the normal dog was greatly disturbed: it became apathetic, showed marked hypersalivation and remained quietly in the corner, while the labyrinthectomized dogs played normally. These results confirm those of Sjöberg. His dogs were no longer crane-sick after bilateral labyrinthectomy. These experiments indicate that labyrinthine stimulation is the essential mechanism in the production of motion sickness.

Differential Labyrinthine Ablation Operations: Cipriani and McNally began some experiments with a view to determining what parts of the labyrinth are affected by the forces generated upon the simple swing. Normal frogs and frogs with the optic nerves severed were observed on one of the standard swings for human subjects, a 13-foot two-point suspension swing, motor driven at 15 complete cycles a minute through a total angular displacement of 80 degrees. The most satisfactory method was for one observer to get on the swing and have the frogs on a small table in his lap. The frogs were seen to make compensatory readjustments of the head, body and limbs. Delabyrinthized frogs did not show any similar reactions. All six semicircular canals were removed from three different frogs; only the two utricles remained intact in these animals. When examined on the tilt table, etc., they gave the usual reactions characteristic of such bisoliutricular frogs; however, each of these three frogs was placed on the swing repeatedly, and none showed the same reactions as did the normal frogs. These experiments are merely suggestive that on the swings a frog with two intact utricles alone is not equipped to respond normally. Is it that the semicircular canals are a necessary part of the mechanism for the reaction to swing stimulation — this in spite of the fact that mathematical calculations tend to exclude angular acceleration and, therefore, semicircular canal stimulation as an important factor in producing swing sickness, etc.? It is hoped to complete these experiments so that well founded

conclusions may be drawn. Such experiments may indicate that the otoliths are not the only part of the labyrinth concerned in the production of motion sickness.

Experiments conducted during the National Research Councils' investigation were regarded as confidential and were published only in committee meeting minutes for restricted circulation. The details of these experiments are now to be published in the general medical press by the experimenters.

CLINICAL TESTS OF OTOLITHIC FUNCTION.

Jones and Fisher, in 1918, described a tilt table on which the patient stood erect. A slow tilt stimulated the labyrinths. The results were unsatisfactory and hard to interpret.

Grahe constructed a special tilt table to which the patient could be securely strapped. There was a double frame so that the whole body could be turned about a vertical or a horizontal axis. From experiments on this table he described tests of otolithic function.

Appreciation of the Vertical: One test is what he called "Appreciation of the Vertical." The patient is placed on the tilt table, eyes closed, head and body fixed, and the table is slowly tilted about a horizontal axis. The patient is instructed to say when the tilting brings his body into the erect position in the vertical plane. The tilting is begun with the patient well out of the vertical plane, either forwards or backwards or to either side. Grahe reported that one's appreciation of the erect position during forward and backward tilting (sagittal plane) is very unreliable. The tilting from side to side (the frontal plane) is more exact. He stated that one usually thinks that the erect position is reached a little before it actually has been.

Compensatory Head Movements: A second test he described is to remove the head holder and repeat the tilting as above. The eyes should be closed in all these tests, to exclude visual reflexes. The patient's head is then watched to see if any compensatory movements of the head occur. Normally when the patient is tilted, for instance, to the left the head should incline to the right shoulder in an attempt to remain erect.

Abnormal Head Positions: The third test he described is to observe the patient while he is standing erect. The patient

remains with the eyes closed and head free to move. Sometimes a spontaneous deviation of the head to either side will occur.

Grahe stated that the deep sensibilities and neck reflexes play a part in these reflexes. The tilting must be carried out very smoothly. If all precautions have been taken to exclude extraneous stimuli, and wide deviations from the normal still occur, one may conclude that either labyrinth shows a failure of its positional reflex mechanism (utricle). These tests have never attained wide application, presumably because information about the otolithic mechanism is still so scanty, but these experiments of Grahe's are probably the best clinical tests yet described for otolithic function.

SUMMARY.

1. The saccular otolithic organ is not part of the equilibrial mechanism.

2. The utricle is the end-organ responsible for otolithic reflexes.

3. a. The utricle is stimulated by: *1.* The forces of gravity; *2.* centrifugal force; *3.* linear acceleration in any direction; *4.* angular acceleration about a horizontal axis.

b. It is not stimulated by angular acceleration about a vertical axis.

4. The semicircular canals as well as the utricles are concerned in the maintenance of postural tone.

5. a. There are no confirmed experiments reported to prove that stimulation of the utricle produces nystagmus.

b. There are no necropsy examinations reported to show that a diseased utricle has been responsible for positional nystagmus or dizziness.

6. a. The manner of utricular stimulation (its propriation) is not understood.

b. There is some evidence to show that the utricle is not constantly signaling. It does not signal at rest but only during head movements.

7. The evidence that it is stimulation of the labyrinth which is responsible for the symptoms of motion sickness is as follows:

a. Deafmutes with nonfunctioning labyrinths are not susceptible to motion sickness.

b. Dogs lose their susceptibility to motion sickness after bilateral labyrinthectomy.

8. The evidence that it is linear acceleration which stimulates the labyrinths and elicits the symptoms of motion sickness is as follows:

a. The forces acting during movements of a ship at sea have been estimated and analyzed by a few observers, and they have concluded that for the most part values of angular acceleration are below the computed threshold of labyrinthine stimulation, whereas the values for linear acceleration are well above the computed labyrinthine threshold.

b. Of the forces acting on a simple two-point suspension swing, the radial (linear) acceleration is the only one which greatly exceeds the computed threshold of labyrinthine stimulation.

9. The evidence that motion sickness results from utricular stimulation is as follows:

a. Individuals suffering from motion sickness, as a rule, do not have nystagmus.

b. The weight of experimental evidence has shown that the otolithic organs are stimulated by linear acceleration.

c. It has been assumed (conclusion 8) that excessive linear acceleration elicits the symptoms of motion sickness.

10. Some experiments indicate that the amount of g (linear acceleration) is not sufficient in itself to elicit the symptoms of motion sickness.

11. More exact measurements (accelerometer measurements) of the forces acting during sea and air sickness are necessary before it can be concluded that linear acceleration is all-important and that angular acceleration can be excluded as a factor.

12. Uncompleted experiments indicate that the semicircular canals may be a factor in producing swing sickness. Differential ablation experiments on the labyrinth are necessary

before it can be concluded that the utricle is the only part of the labyrinth concerned in the production of motion sickness.

13. Adequate clinical tests of utricular function are still lacking.

14. Great restraint is needed in drawing conclusions about utricular function from poorly controlled clinical experiments. There are many extralabyrinthine factors which may be playing a part and which are difficult to assess and exclude.

For the complete bibliography exclusive of the experiments conducted during the National Research Councils' investigations, the reader is referred to the review by McNally and Stuart.

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PNEUMATIZATION OF THE ADULT TEMPORAL BONE, THE MASTOID PORTION. AN ANATOMIC AND CLINICAL STUDY.*

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In recent years many articles have appeared in the literature on the subject of pneumatization of the petrous portion of the temporal bone; but, during the same period, there has been little published on pneumatization of the mastoid portion of the temporal bone, yet we continue to see many deafened patients with chronic suppuration of the middle ear and mastoid, and not infrequently patients with intracranial complications resulting from incomplete mastoid surgery. In view of the fact that most of the mastoid cell groups can be completely and easily exenterated surgically, one must conclude that the operator who fails to eradicate diseased cell areas in the mastoid does not have a thorough knowledge of the origin, course, distribution and important relations of the various cell groups in this area. It is with the above thoughts in mind that I offer this discussion.

MATERIAL.

The material for this study consists of 100 gross dissections from 52 adult skulls. In doing the dissections, regular mastoid gouges were used to remove the outer cortex over the mastoid. The remainder of the work was done with an electric motor and dental burs. Tracts leading from the mastoid into the petrous pyramid were uncovered and followed by removing the cortex from the anterior and posterior surfaces of the petrosa in a plane parallel to the plane of the superior semicircular canal adjacent to and anteromedial to the canal. No effort was made to expose and follow tracts into the pyramid from the tubal and infralabyrinthian areas. Numerous

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illustrations are used to show the various tracts, cell groups and other important anatomic relations.

ANATOMY.

A brief review of the gross anatomy of the temporal bone will help to understand more clearly the extent, location and relations of the various cell groups that may be present. It will aid in the interpretation of symptoms and physical signs that may occur in the presence of mastoid infection, in ing complications and in selecting the logical surgical appro locating complications and in selecting the logical surgical approach to the areas that may be involved.

The temporal bone is made up by the fusion of three distinct parts — the squama, the annulus and the petrosa. The three parts are clearly visible on the surface of the skull where complete fusion between the squama and the mastoid portion of the petrosa has failed to occur (see Fig. 1).

Relations of the Temporal Bone: The fused bone articulates superiorly with the parietal bone, anteriorly with the greater wing of the sphenoid and the maxilla, inferiorly with the mandible, medially with the body of the sphenoid and posteriorly and posteromedially with the occipital bone.

The greater portion of the outer surface of the squama is covered with the temporal muscle. Its inner surface is in contact with the dura of the middle fossa. On this surface the main branch of the middle meningeal artery courses forward, outward, upward and then curves backward.

The sternomastoid muscle is attached to the outer surface of the petrous portion of the mastoid process, and at a deeper level the digastric muscle is attached along the digastric groove. More posteriorly is found the foramen for the mastoid emissary vein. The inner surface of the mastoid portion of the petrous bone is in contact with the lateral sinus and the dura of the posterior fossa posterior to the sinus. Anteriorly this surface is continuous with the posterior surface of the petrous pyramid.

The Mastoid: The mastoid portion of the temporal bone has a dual origin. The squama forms its anterosuperior and superficial portion; it forms the outer half of the tegmen and mastoid antrum, all of the superior external bony canal wall,

most of the posterosuperior canal wall, and the cortex over the anterosuperior portion of the mastoid.

The petrosa forms the posteroinferior and deep portions of the mastoid including all of the mastoid tip, and the inner half of the tegmen and antrum; it articulates posteriorly and

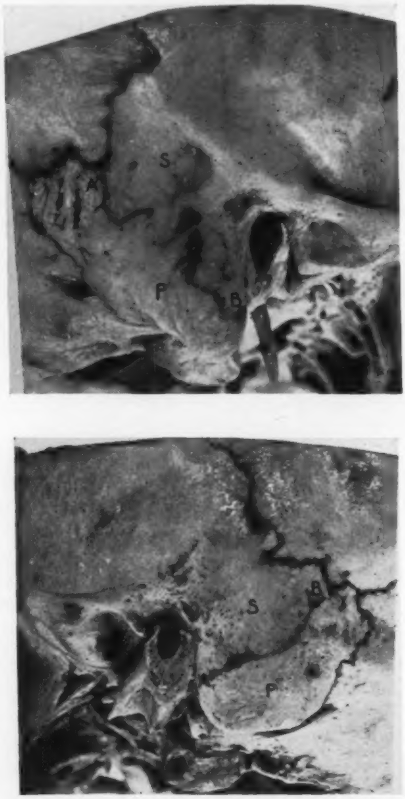

Fig. 1. Persistent squamopetrosal suture (AB), (S) squama, (P) petrosa.

posteroinferiorly with the occipital bone; here the two bones are often fused. Anteromedially it is continuous with the base of the petrous pyramid. The facial nerve courses backward through the middle ear beneath the overhanging ledge

of the horizontal semicircular canal to enter the deep portion of the posterior bony external auditory canal. Immediately after entering the posterior canal wall, the nerve curves downward to assume its vertical course to the stylomastoid foramen; at this point the nerve lies directly medial to the tympanomastoid suture at its most inferior portion; superiorly the suture lies anterior to the nerve. A vertical line extending superiorly from the lower portion of the tympanomastoid suture accurately locates the anterior-posterior position of the nerve. The deep position of the nerve is located by a vertical line extending from beneath the overhang of the horizontal semicircular canal to the anterior end of the digastric ridge.

PNEUMATIZATION.

Pneumatization of the adult mastoid may be confined to the mastoid antrum or it may extend extensively into the squama, all of the petromastoid, and occasionally cells may cross a fused petrooccipital suture to invade the occipital bone. Often tracts extend from the mastoid into the petrous pyramid. Between and including the above two extremes are many unusual and interesting variations.

In this discussion cells confined to the squama will be designated as squamosal cells. Cells in the petromastoid will be referred to as petrosal cells and are not to be confused with cells in the deeper portions of the petrous pyramid; cells in the posterior portion of the petromastoid along the occipitomastoid suture will be referred to as marginal cells, those above the emissary vein will be called superior marginal cells, those below the vein will be termed inferior marginal cells. Since the cells in the base of the petrous pyramid external to the superior semicircular canal and those inferior to the posterior semicircular canal are usually involved in acute suppuration of the mastoid and are best dealt with through the mastoid approach, they will be considered as a direct continuation of the mastoid cells.

Classification of Cells: On an anatomic basis the mastoid cells, frequently separated by septa or isolated by tracts, are

divided into two primary groups with several subgroups as follows:

I. *Squamosal Cells:*

1. Posteroinferolateral cells.
2. Cells on undersurface of tegmen.
3. Superior cells.
4. Anterior cells.

1. Zygomatic cells.
2. Suprazygomatic cells.
a. Secondary inner surface cells.
3. Primary inner surface cells.

II. Petrosal Cells:

1. Inferior cells.
1. Superficial cells.
2. Deep cells.
a. Cells to deep portion of mastoid process.
b. Cells medial to digastric ridge.
c. Retrofacial and infralabyrinthian cells.

2. Superior cells.

1. Superoposterior cells (sinodural angle cells).
2. Cells on lower surface of tegmen.
3. Solid angle cells.

I. Squamosal Cell Group: 1. Posteroinferolateral Cells: This cell group arises from the inferolateral part of the squamosal portion of the antrum. They lie immediately beneath the squamosal cortex lateral to the antrum, are in contact with the posterosuperior canal wall, and extend posteroinferiorly to the petrous portion of the mastoid.

2. Cells on the Undersurface of the Tegmen: These cells arise from the upper squamosal portion of the antrum; they are small, compact cells tightly adherent to the tegmen.

3. Superior Cells: These cells arise from the superior squamosal portion of the antrum and extend upward, often to a high level, above the tegmen; they may course forward

into the posterior root of the zygoma or they may pass above the zygoma as suprazygomatic cells (see Figs. 2 and 3).

4. Anterior Cells: There are two groups of anterior cells: *1.* zygomatic cells which terminate in the posterior root of the zygoma, and *2.* suprazygomatic cells. These latter cells extend above the zygoma and may course forward almost to the anterior border of the squama; they may extend forward above the external auditory canal in successive groups of

Fig. 2. Superior squamosal cells terminating in the posterior root of zygoma.

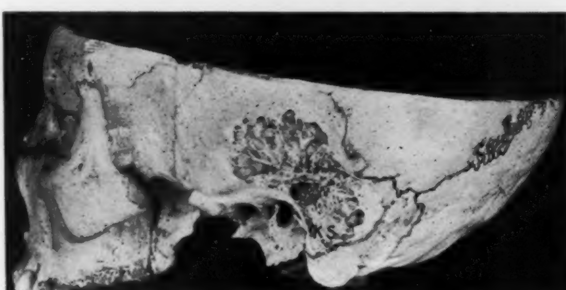

Fig. 3. Superior squamosal cells extending forward above zygoma as suprazygomatic cells.

fairly large cells or they may penetrate rather denser bone in the form of numerous small channels and dilate into well defined cells more anteriorly (see Figs. 4, 5 and 6). Cells of this group may dip downward into both glenoid tubercles, may extend deeply inward between the inner table and the anterior root of the zygoma and may even extend into the

proximal portion of the zygomatic arch (see Fig. 7). Often cells of this group lie immediately above the temporomandibular joint and are separated from the joint cavity by bone only a small fraction of a millimeter thick. Cells from this group occasionally penetrate the inner table through tiny channels to reach the inner surface of the squama in the bend

Fig. 4. Suprazygomatic cells extending forward in successive groups almost to anterior border of squama. Note extensive pneumatization throughout entire mastoid area.

Fig. 5. Cells have extended forward via small channels through dense bone (arrow) and have dilated into large cells (C).

of the middle meningeal artery; these cells are designated as secondary inner surface cells (see Fig. 8). Another group of cells found in the same locality, but less often, originate from the most anterior portion of the antrum or from the

epitympanum and course outward, upward and forward to their termination; these cells have no connection with other mastoid cells and are designated as primary inner surface cells (see Fig. 9).

When the squamosal cells are carefully removed, a complete or partial septum is frequently found, below the antrum,

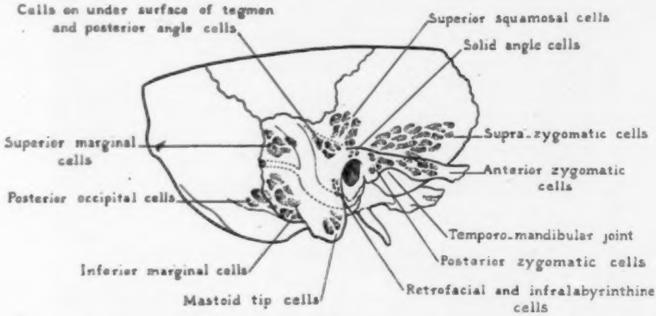

Fig. 6. Semidiagrammatic illustration showing many of the cell groups found in a well pneumatized mastoid; compare with Fig. 4.

Fig. 7. Cells in proximal portion of zygomatic arch.

separating the squama from the petrosa. This septum is known as Korner's septum (see KS, Fig. 3).

II. Petrosal Cell Group: 1. Inferior Cells: The inferior cells are divided into two main groups: *1.* the superficial cells, and *2.* the deep cells. The superficial cells arise from the outer part of the petrosal portion of the mastoid antrum.

Fig. 8. Secondary inner surface cells; bristles are in channels leading from mastoid; note relation of cells to groove for middle meningeal artery.

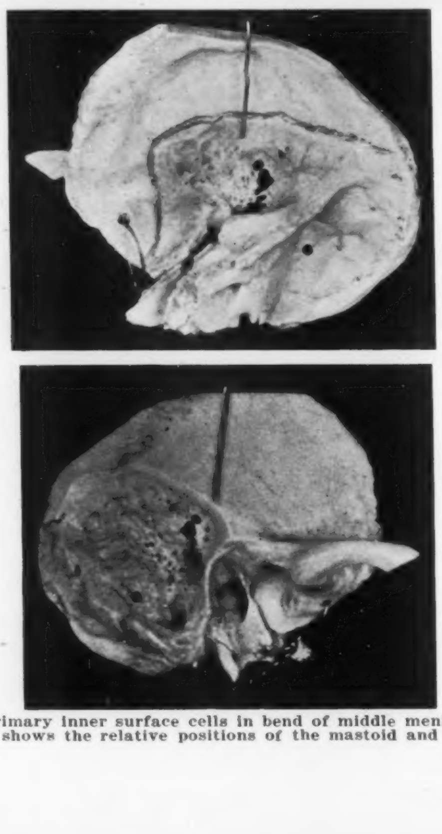

Fig. 9. Primary inner surface cells in bend of middle meningeal artery; the marker shows the relative positions of the mastoid and inner surface cells.

They occupy all of the superficial part of the mastoid process; in addition they extend posteriorly, external to the upper portion of the sigmoid sinus and above the emissary vein, often reaching the occipitomastoid suture; these are the superior marginal cells. Frequently there is an unnamed septum separating the superficial cells from the deep petrosal

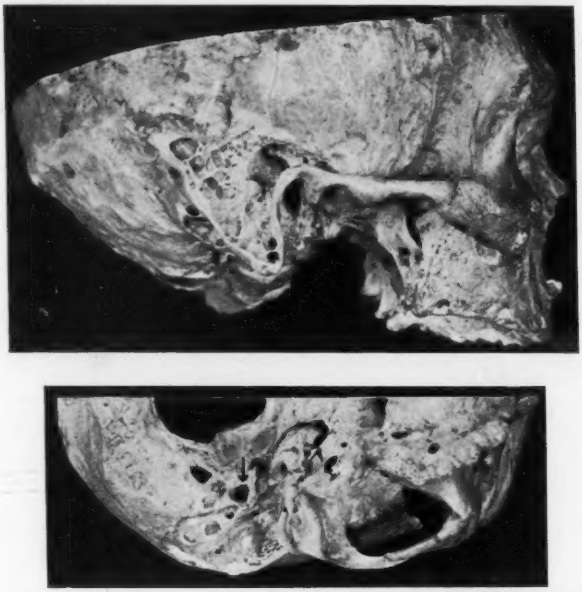

Fig. 10. Lateral view shows unnamed septum separating the superficial petrosal cells from the deep cells; inferior view shows deep mastoid cells and basioccipital cells.

cells (see Figs. 10 and 11). The deep petrosal cells arise from the deep portion of the antrum from a common tract or from separate tracts in the form of small extended channels, and descend posterior and parallel to the facial canal to form the deep cells of the mastoid process, the cells medial to the digastric ridge, and the retrofacial and infralabyrinthian cells. These various cell groups are often separated by dense septa. In their downward course, small channel-like cells may extend forward from the deep mastoid cells and the cells medial to the digastric ridge to surround or almost surround the facial canal. Inferiorly the cells just medial

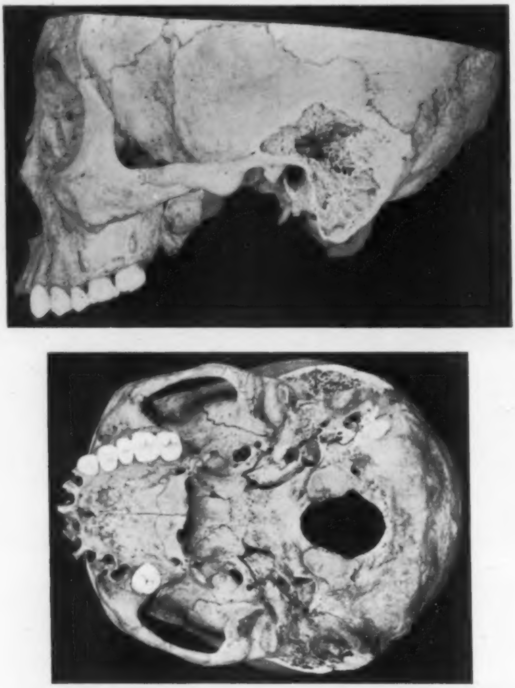

Fig. 11. Lateral view shows septum separating superficial cells from deep cells; inferior view shows deep mastoid cells and basioccipital cells with bristle passing upward beneath septum into antrum.

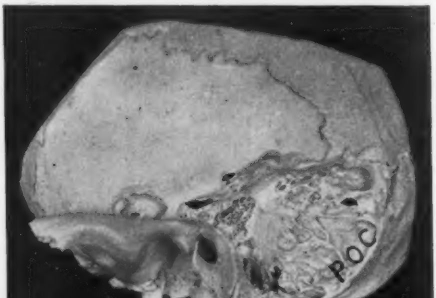

Fig. 12. Inferior marginal cells that have crossed a fused suture line to invade the occipital bone as posterior occipital cells (POC).

to the digastric ridge often extend beneath the inferior knee of the sigmoid sinus toward the jugular bulb; other cells of this same group extend posteriorly and superiorly along the posterior border of the descending portion of the sigmoid sinus to occupy the area beneath the emissary vein, these are the inferior marginal cells; these cells may cross a fused

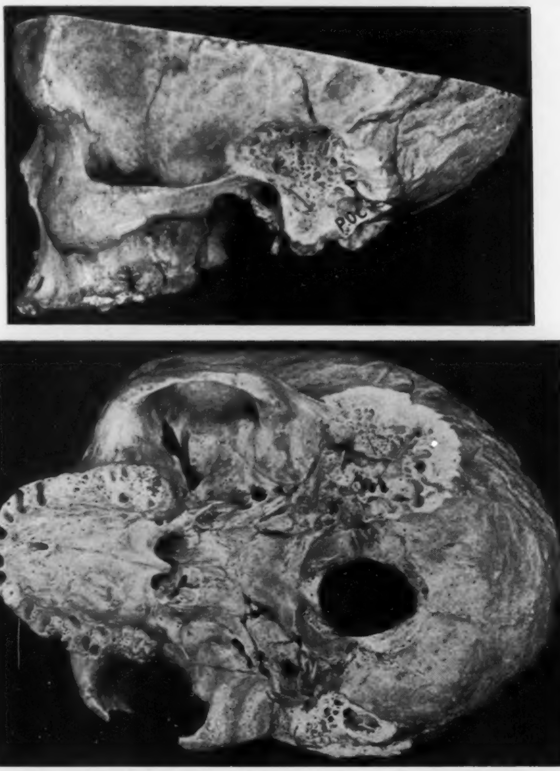

Fig. 13. Lateral view shows extension of cells from superficial cell group into occipital bone (POC), and a septum separating superficial cells from deep cells; the bristle shows small tract connecting with deep cells; inferior view shows deep mastoid cells and basioccipital cells.

occipitomastoid suture to invade the occipital bone (see Fig. 12). Occipital cells in this locality will be called posterior occipital cells. Infrequently, cells extend from the superficial petrosal cell group to the area beneath the emissary vein (see Fig. 13). The emissary vein may pass directly through

the mastoid cavity separating the superior marginal cells above from the inferior cells below (see Fig. 14). The retrofacial and infralabyrinthian cells arise from the deepest inferior portion of the antrum and extend downward and inward to their destination below the posterior semicircular

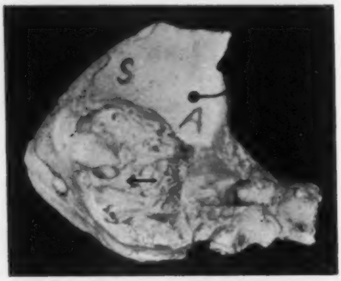

Fig. 14. Large emissary vein in mastoid cavity separating superior marginal cells from inferior marginal cells.

Fig. 15. Shows bristle in tympanomastoid tract deep to facial nerve.

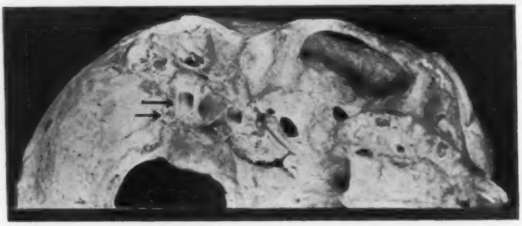

Fig. 16. Inferior view showing basioccipital cells extending to articular facet of occipital bone.

canal and adjacent to the jugular bulb; occasionally these cells arise from the middle ear cavity and pass backward medial to the facial nerve to reach the same location (see

Fig. 15). Cells from this group may cross a fused petro-occipital suture and pass posteroinferomedially beneath the terminal portion of the sigmoid sinus to invade the basilar portion of the occipital bone and will be designated as basi-occipital cells; they may terminate just short of the foramen magnum (see Figs. 16 and 17).

2. Superior Cells: The superior cells are subdivided into three groups: *1.* superoposterior cells or sinodural angle cells; *2.* cells on lower surface of tegmen; and *3.* solid angle

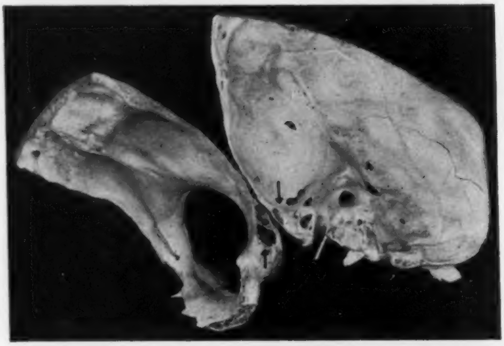

Fig. 17. Shows extension of basioccipital cells beneath terminal portion of sigmoid sinus reaching to foramen magnum.

cells. The superoposterior cells (sinodural angle cells) occupy the sinodural angle and the angle between the posterior and anterior surfaces of the petrous pyramid; the outer cells of this group course upward, backward and outward to reach the cortex beneath the posterior-inferior border of the squama. Tracts may extend from the inner cells of this group into the deeper portions of the petrous pyramid; such tracts may extend between the posterior surface of the pyramid and the posterior semicircular canal or in the angle between the posterior and anterior surfaces of the pyramid. The cells on the lower surface of the tegmen are small, compact cells tightly adherent to the tegmen; tracts from these cells may extend into the petrous pyramid above or anterior to the superior semicircular canal. The solid angle cells are situated in the angle formed by the three semicircular canals. A tract may extend from these cells through the arch of the superior semicircular canal into the petrous pyramid.

Table showing the mastoids grouped according to the extent of pneumatization present. The frequency with which cell groups extending beyond the normal boundaries of the mastoid, and cell tracts leading from the tympanum into the infralabyrinthian cells of the mastoid and tracts extending from the mastoid into the petrous pyramid are also included.

	Extensively Pneumatized Mastoids, 33	Well Pneumatized Mastoids, 37	Moderately Pneumatized Mastoids, 22	Non-Pneumatized Mastoids, 8	Total 100
Superior squamosal cells	15	8	0	0	23
Suprazygomatic cells	18	0	0	0	18
Secondary inner surface cells	8	0	0	0	8
Primary inner surface cells	0	2	0	0	2
Posterior occipital cells	4	2	0	0	6
Basioccipital cells	5	3	0	0	8
Infralabyrinthian cells	30	29	5	0	64
Tympanomastoid tracts	4	3	2	0	9
Tracts from mastoid into petrous pyramid	16	4	0	0	20

CLINICAL CONSIDERATIONS.

In a typical case of acute surgical mastoiditis there are several classical symptoms and physical findings on which a diagnosis is made. Briefly those symptoms and physical findings are:

1. Pain in the ear and mastoid, worse at night.
2. Deafness, conductive in type.
3. Fever.
4. Edema and redness over the mastoid.
5. Mastoid tenderness:
   - *a.* Over antrum.
   - *b.* In region of emissary vein.
   - *c.* Over tip.
6. Discharge from middle ear.
7. Edema with sagging of posterosuperior bony canal wall.

In evaluating the symptoms and physical findings associated with acute infection of the mastoid, it is imperative that one keep in mind modifications of the typical case as a result of infection in remote cell areas. One must also consider the altered clinical picture that results when infection becomes localized and confined to a definite cell group by some of the various septa that have been described, or because of the small extended channels through which certain cell groups are connected with the antrum. Edema with constriction of the posterosuperior bony external auditory canal wall is the most reliable of all early physical signs in making a diagnosis of acute surgical mastoiditis, but this manifestation of disease can occur only when there is active involvement of the posteroinferolateral cell group of the squamosa. Infection in other parts of the mastoid does not and cannot produce sagging of the posterosuperior bony canal wall.

Infection in the suprazygomatic cells is associated with pain and tenderness in this area; if there are cells immediately overlying the temporomandibular joint there will be pain and tenderness in the joint, and chewing or opening the mouth will accentuate the pain. Infection may involve the joint cavity by erosion and perforation of the overlying bone; infection in the primary inner surface cells may also produce temporomandibular joint symptoms and complications. Erosion of the outer cortex will produce a subperiosteal, subtemporal abscess anterior to the auricle; with such a complication, pain on chewing is a prominent symptom. Erosion of the inner plate will involve the structures in the middle fossa. When infection becomes localized to a definite area it is obvious that all localizing signs and symptoms will point to the area involved.

Infection in the inferior deep petrosal cells produces deep-seated pain in the mastoid. If tenderness is present, it will be found only on the deep surface of the mastoid tip and over the area at the posterior end of the digastric groove beneath the emissary vein. Infection in the deeper cells of this group, namely, the retrofacial, infralabyrinthian and basioccipital cells, produces deep-seated pain or headache but no surface tenderness because of its deep location. A Bezold's abscess is the most common external complication following

infection of the deep petrosal cells, but erosion with abscess formation may occur beneath the emissary vein or at any point medial to the digastric groove inward to the vertebral column. Intracranial extensions may involve the sigmoid portion of the lateral sinus or the other structures of the posterior fossa; the latter may be involved posterior to the sinus or anteriorly in the region of Trautman's triangle. Infection in this cell group may also involve the facial nerve and the inner ear.

Roentgenographic Examination: Persistent pain in any portion of the mastoid area following an acute upper respiratory infection associated with even the slightest middle ear symptoms, either with or without drainage, should lead one to suspect infection in the mastoid. In all such cases, and in all cases of frank mastoiditis, a thorough Roentgenographic examination of the mastoid area is indicated. Such examination should include a lateral view, an anterior-posterior view and the occiput down view. The examination should be repeated if necessary. In addition to its diagnostic value, much valuable anatomic information is secured by such examination. In making the lateral view, the exposure should be centered over the mastoid antrum and should extend to include the occipitomastoid suture posteriorly and the anterior border of the squama anteriorly. The lateral view will show:

1. The extent of pneumatization in the squama and petromastoid; and the relation of the various cell groups to the tegmen, the external auditory canal, root of the zygoma, temporomandibular joint, and to the parietomastoid and occipitomastoid sutures.

2. The size and location of the sigmoid sinus.

3. Size and position of emissary vein.

4. Large diploic veins if present.

The anteroposterior view will show the extent of pneumatization of the mastoid tip, while the occiput down view will show the infralabyrinthian cells that lie beneath the posterior semicircular canal; this view will also show the basioccipital cell extensions from the infralabyrinthian cells. Surgery on the adult mastoid should never be resorted to until a thorough Roentgenologic study is made.

Surgical Approach: After all preliminary studies have been completed and it is apparent that the mastoid should be operated upon, the first problem is the incision of the overlying soft tissues. If there is widespread pneumatization of the squama extending high and almost to the anterior border, associated with cells extending posteriorly to the occipitomastoid suture, it is apparent that the incision should be started more anteriorly and at a higher level than usual; the incision should swing well backward, downward and then forward to secure the exposure needed.

If the above-mentioned cell arrangement is present, one knows that there is a cushion of cells overlying the inner table of the squama above the tegmen, and that a similar layer of cells separates the outer cortex from the sinus plate and the inner table posteriorly; hence, it is perfectly safe to start at the upper level of the squamosal cells and remove all of the outer table over the entire mastoid, using a large gouge. After this is done the mastoid antrum is approached by following the course of the posterosuperior portion of the external bony canal wall.

The suprazygomatic cells are best removed from the mastoid approach using curettes and rongeuers of suitable size. In rare cases where cells stop just short of the anterior border of the squama, it may be feasible to split the temporal muscle vertically and approach the cells directly; this is especially true if intracranial complications have developed in this region. In approaching the inner surface cells in this area, both the primary and the secondary, it is necessary to remove the inner table through the mastoid wound and approach them from behind; the small channels previously mentioned in connection with the secondary cells will lead one to this group. Good Roentgen films will aid one in locating the primary inner surface cells. In the removal of these cells one must be careful to avoid injury to the middle meningeal artery.

The removal of the inferior superficial petrosal cells should give no trouble, but frequently the marginal cells of this group are left in the posterosuperior portion of the mastoid beneath the cortex, in the angle formed by the parietomastoid and occipitomastoid sutures.

The deep cells of the mastoid process lie deep to the superficial petrosal cells and may be separated from them by a rather dense solid septum; when such a septum is present the cells are reached by working downward and outward from the mastoid antrum with a curette of suitable size. Removal

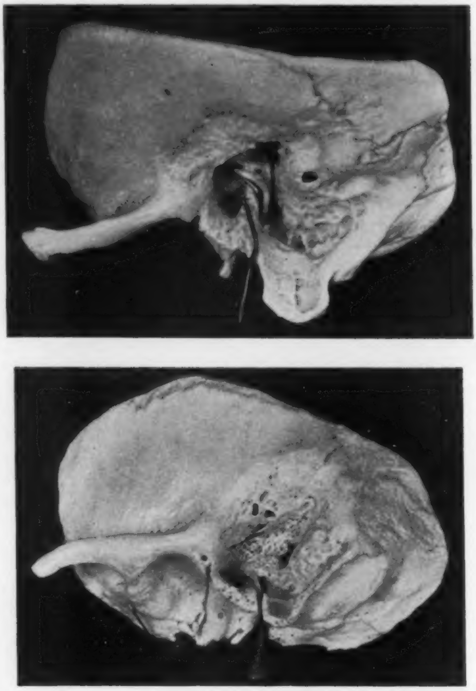

Fig. 18. The facial canal has been opened and its relations to the tympanomastoid suture, retrofacial cells and the horizontal semicircular canal are shown.

of the deep mastoid cells brings the operation down, or in, to the level of the facial nerve and the digastric ridge.

Cells medial to the digastric ridge, the retrofacial cells, and the infralabyrinthian cells with their occipital extension still remain to be exposed and exenterated; tracts to these various cell groups pass downward from the deepest portion of the antrum between the deep portion of the external auditory canal wall and the descending portion of the sigmoid sinus

and deep to the facial nerve; in this position the various tracts are often covered with a dense septum, which is best removed with a small gouge (3 mm.), working from above downward and keeping close to the anterior border of the sigmoid sinus, thus avoiding the facial nerve. After the vari-

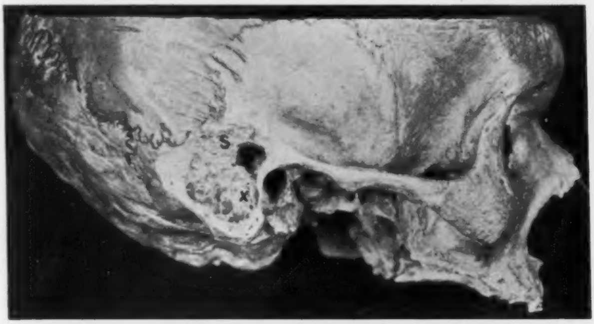

Fig. 19. Here the (sinodural angle) cells are hidden beneath a septum (S); below, there is a septum overlying the retrofacial cells (X).

ous cell tracts have been opened it is a simple procedure to complete the removal of the various cell groups. The basi-occipital cells are a continuation of the infralabyrinthian cells and can be followed and exenterated without difficulty. When these cells are removed the inferior wall of the sigmoid sinus is exposed throughout its entire length. Experienced operators are sometimes confused when they first encounter this type of cell arrangement. Injury to the facial nerve can be avoided by working posterior to a vertical line drawn upward from the lower portion of the tympanomastoid suture, and lateral and medial to a vertical line drawn from the overhang of the horizontal semicircular canal to the anterior end of the digastric ridge with the patient's head placed in a horizontal position in the saggital plane (see Fig. 18).

The superior petrosal cells lie above the antrum and are not difficult to expose or exenterate. All or part of the supero-posterior group (sinodural angle cells) may lie beneath a septum and are often overlooked (see Fig. 19). When the three cell groups in this area are completely removed to the inner ear capsule, tracts leading from their inner surfaces into the deeper portions of the petrous pyramid are often exposed.

THE NONPNEUMATIC MASTOID.

In this discussion little need be said regarding the nonpneumatic mastoid. When pneumatization fails to occur, the overlying cortex is thick, the sigmoid sinus may encroach upon the antrum and the posterior bony external auditory canal wall, and the tegmen may dip downward posteriorly and laterally to overhang the antrum. In such a mastoid care must be exercised, in approaching the antrum surgically, not to injure the dura or the sinus.

COMMENT.

At birth pneumatization of the mastoid is limited to the antrum; but under normal conditions growth of the various tracts and cell groups is rapid and by the end of the sixth year or soon thereafter full adult development has occurred.

In the process of pneumatization, there is usually free intercommunication between the squamosal and petrosal cells, and between adjacent subgroups of cells; however, not infrequently cell groups may be more or less completely isolated by extended channel-like tracts or by rather dense septa and the clinical importance of such isolation in the presence of infection is obvious. Failure to seek out and exenterate such cell groups at operation will result in prolonged drainage, and may give rise to serious or fatal complications.

CONCLUSIONS.

1. Operation upon a pneumatic mastoid should never be done until thorough Roentgenologic studies have been made.

2. An exact knowledge of the origin, course, distribution, final termination and important relations of all the various mastoid cell groups will enable the aural surgeon to follow and exenterate them with safety and assurance.

ACHALASIA OF THE CARDIA (CARDIOSPASM). THIAMIN CHLORIDE AS ADJUNCT IN TREATMENT. PRELIMINARY REPORT.*

PAUL M. MOORE, JR., M.D.,
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The condition known as cardiospasm, and more recently as achalasia of the cardia, has been recognized and treated since Thomas Willis reported the first case in 1672.[59] Numerous theories have been advanced as to its etiology and pathology, but in the main they may be grouped into two opposing schools.[43,44] Until recently, greatest credence was given to the theory that cardiospasm is due to spasm of the lower end of the esophagus. Meltzer[39] and Mikulicz[40] attributed the condition to spasm of the cardiac sphincter and named it "cardiospasm," which has been the most common term for the disease. Jackson[27] advanced the theory that it is due to spasm of the crura and called it "phrenospasm." Between these two groups is Mosher's[41] interpretation, fibrosis of the terminal portion of the esophagus caused by infection of the walls which produces a tubular narrowing.

The second theory was first advanced by Einhorn[12] in 1888, again by Rolleston[34,48] in 1896, and independently by Hurst[23] in 1915. They attributed the condition to failure of the cardiac sphincter to open during swallowing and opposed the idea that actual spasm exists. Hurst proposed the name of "achalasia of the cardia" for the disease. This idea has been gaining in popularity recently and has many points in its favor. A similar hypothesis was offered by Hill,[22] who believed that the crura of the diaphragm fail to relax.

PHYSIOLOGY.

A better understanding of the pathology of the disease can be achieved if one keeps in mind the normal physiology of the esophagus. Normally, swallowing is initiated by voluntary movement of the mouth, tongue and pharynx.[10,21,25,38,44,55,56] The involuntary phase occurs when the bolus reaches the

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hypopharynx. At this point a peristaltic wave starts, passes through the cricopharyngeus and proceeds down the esophagus to the cardia; here there is a slight pause, the cardia relaxes and the bolus passes into the stomach. The active peristaltic wave is preceded by a wave of relaxation which permits the cricopharyngeus muscle and the cardia to open and allows the bolus to distend the esophagus easily in passing. This is known as the primary peristaltic wave.

A secondary peristaltic wave sometimes is seen, which starts in about the midesophagus and progresses into

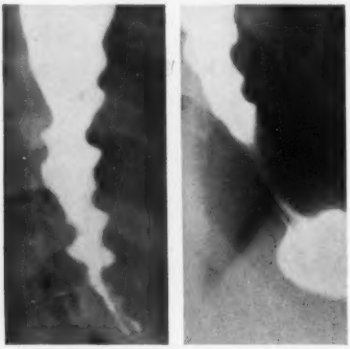


Fig. 1. Local segmental contractions in a case of cardiospasm.

the stomach. Local segmental contractions are occasionally encountered (see Fig. 1). These are sudden tonic contractions of a portion of the esophageal wall, which do not progress downward but remain a moment and then relax. They are not necessarily associated with any symptoms, although they may produce cramp-like pains. It may be that they are the cause of the severe pains found in some cases of achalasia of the cardia.

In a definite case of achalasia the primary peristaltic wave passes through the cricopharyngeus and disappears.[56] The esophagus is a distended bag, which fills with food and liquids to a certain level before the cardia opens to allow the passage of some of its contents. Although Hurst[23,25] placed this level at 8 inches, we have noted a variation in its height in different patients. The flow into the stomach is slow and

ceases when the level of the material in the esophagus reaches its former height. Many writers mention the occurrence of strong peristaltic waves in the distended hypertrophied esophagus, but they fail to describe these waves; however, Hurst in a report of a case of seven years' duration speaks of observing "extraordinarily vigorous peristaltic waves passing down to the cardia."[23] The waves usually seen are local segmental contractions which do not progress down the esophagus; they remain stationary, force the contents upwards as well as downwards and then relax. They are not purposeful, useful waves but the result of a disturbed neuromuscular reaction.

ANATOMY.

The normal esophagus is a muscular tube 23 to 24 cm. in length extending from the hypopharynx to the stomach. Its walls are composed of four layers: *1.* the external fibrous, *2.* the muscular, consisting of an external longitudinal and an inner circular layer, *3.* the submucous coat, and *4.* the inner mucous coat. The normal thickness of the walls is about 1.5 mm.[49] The cricopharyngeus muscle acts as a sphincter at the upper end.

There has been much discussion concerning the cardiac end of the esophagus. In the adult there is no well defined sphincter muscle, but in the stillborn the abdominal portion shows a muscular coat distinctly thicker than the rest of the esophagus.[23] The abdominal portion acts as a sphincter to prevent regurgitation of food and gas into the esophagus from the stomach. Normally it is closed, opening only for the passage of food, or if the intragastric pressure becomes too great there may be eructation or vomiting. It is usually stated that the muscular fibres of the upper portion of the esophagus are striated, those of the middle portion are mixed and those of the lower portion are smooth; however, striated fibres may appear in the lower third. The nerve supply is parasympathetic via the vagus and sympathetic from the celiac plexus via the fibres which follow the left gastric artery.[13,31] Ganglion cells, similar to Auerbach's plexus, are situated between the circular and longitudinal muscular coats. These are more numerous near the cardia.[29]

PATHOLOGY.

In achalasia of the cardia there is first a marked hypertrophy of the circular muscle coat, which is thought to be due to an attempt on the part of the esophagus to overcome obstruction to the passage of food through the cardia. Russell,[49] in 1898, reported the esophageal wall to be thickened to 1 cm. Later there is a marked dilatation of the lumen of the esophagus, which Hurst and Rake[25] believe is due to destruction of the ganglion cells and subsequent loss of vagus innervation. The dilatation may become enormous in advanced cases, and as much as 2,800 cc. of fluid has been aspirated from one patient.[17] In the far advanced case the esophagus may become so lengthened that it sags to the right of the cardia and forms a large sac resting on the dome of the diaphragm. This acts as a reservoir for stagnating food and probably never empties completely.

Involvement of the ganglion cells of Auerbach's plexus has been reported by several writers.[2,7,14,24,25,36,41] This consists of inflammatory changes followed by loss of the cells and eventually by a thin scar. Both Mosher[41] and Freeman[18] reported one case of long standing without involvement of the ganglion cells. While the muscular coat of the esophagus is greatly hypertrophied, hypertrophy has not been reported in the region of the cardiac sphincter. This area remains unchanged. Those who oppose the idea of the disease being due to a spasm of this area point out that if a spasm had existed during life there should be a great hypertrophy of the muscle of this area.

The folds of the mucosa are smoothed out as a result of dilatation of the tube. With retention and decomposition of food, the resultant inflammatory reaction may be severe enough to cause ulceration. Carcinoma resulting from changes in the macerated mucosa has been reported in a few cases.[4,32,46]

EXPERIMENTAL OBSERVATIONS.

Changes in the esophagus similar to those in achalasia of the cardia have been produced repeatedly by many different workers by sectioning and otherwise interrupting the vagi in cats, dogs, monkeys and rabbits.[9,16,19,30,35,47,61] Langley,[33] in 1899, demonstrated in rabbits relaxation of the cardiac

sphincter on stimulation of the vagi. He first injected curari to paralyze the nerve endings in the striated muscles of the esophagus, and then atropine sulfate to weaken the motor nerve fibres. Without these drugs the esophagus was thrown into violent contraction, which he said effectively blocked the entrance to the stomach. He also referred to an earlier work by Openchowski, in 1883. Stimulation of the sympathetic nerves from the celiac ganglion causes contraction of the sphincter.

These observations indicate the necessity for an intact vagus innervation to the musculature of the esophagus to produce and maintain the normal act of swallowing. They also indicate that disruption of this supply will produce a condition in experimental animals which is similar to cardiospasm, or achalasia of the cardia.

VITAMIN B DEFICIENCY.

Etzel[14] and Stinson[52,53] contend that the primary cause of the disease is a chronic vitamin B_1 deficiency. Both refer to earlier reports along this line by Etzel. They base their conclusions on the similarity between achalasia of the cardia and vitamin B_1 deficiency. Etzel states that megaesophagus, megacolon, pyloric achalasia (pylorospasm), megaloureter, alterations in gastric chemistry and in the electrocardiogram, polyneuritis and low basal metabolic rate are manifestations of degenerative disease of the autonomic intramural nervous system caused by a chronic deficiency of vitamin B_1. He also reports degeneration of Auerbach's plexus and refers to 626 cases from Brazil which showed various combinations of deficiency manifestations. He states that this large number is due to the poor food, low in vitamin B_1, eaten by the lower classes in Brazil. Other writers have reported megaesophagus associated with megacolon, megaloureter and pylorospasm.[11,23,25,26,42,57]

Stinson[53] refers to the marked analogy between avitaminosis B_1 and cardiospasm; anorexia, degeneration of Auerbach's plexus, ptosis of the diaphragm and visceroptosis occur in both. Dilatation of the colon, gastric atony and intestinal stasis occur in B_1 deficiency, and dilatation of the esophagus, esophageal atony and stasis occur in cardiospasm. He reports four cases relieved by intramuscular injections of thiamin

chloride; three of which obtained only partial relief from dilations. One patient was given an injection of 6,000 units of thiamin chloride and was swallowing fluids comfortably in a few minutes. The other three received several injections of 3,000 units at three- to four-day intervals.

Browne and McHardy,[5] in speaking of vitamin B deficiency, state that thiamin chloride may be used to supplement other measures. Marsh[37] reports a case in which both pellagra and cardiospasm existed. Sodeman[51] agrees that there is a vitamin B_1 deficiency in cardiospasm but raises the question whether it is a cause or an effect of the condition.

SYMPTOMS.

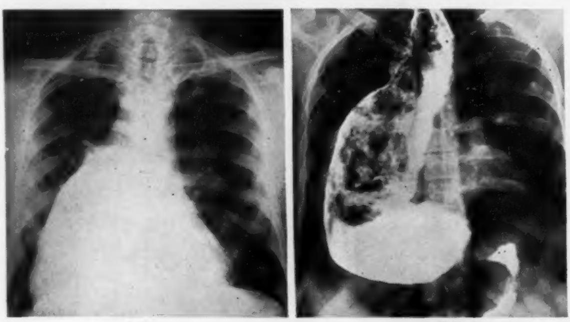
The patient is unaware of the disease until he suddenly experiences a sensation of food sticking momentarily in the lower end of the esophagus. This is usually transitory and may not recur for some time. As the condition progresses, these episodes become more frequent and more severe.

Substernal or epigastric pain and a feeling of pressure often develop. The pain is increased by ingesting food, is sometimes severe and may radiate through to the back. This may be mistaken for an attack of angina. In nervous and excitable patients the pain is more likely to be severe. Regurgitation develops sooner or later, and the regurgitated food has an alkaline reaction, which may show some putrefaction without evidence of digestion. One of our patients complained only of fluid running into her mouth when she stooped over. As the esophagus becomes more and more dilated, shortness of breath develops upon exertion or upon lying down. Severe paroxysms of nocturnal coughing may develop from liquid spilling over into the larynx. Most patients attain some comfort by voluntarily regurgitating the food left in the esophagus after a meal. Drinking warm water, exhaling completely or twisting the body aid in the passage of the food into the stomach. The majority of these people eventually reach a balance where their weight is maintained at a certain level. Aside from the embarrassment of trying to eat in the presence of others, and of an occasional need of emptying the esophagus of food, they get along comparatively well. One of our patients, a farmer, maintains a weight of around 200 pounds, although he has a huge esophagus with a sacculated lower end, which

probably never empties completely. Other cases of long-standing may lose weight until they become very emaciated. Some patients develop marked toxemia and a septic temperature as the result of inflammation of the esophagus due to the long retention of decaying food.

DIAGNOSIS.

The diagnosis usually can be made readily from Roentgenograms of the esophagus. On the flat chest plate a markedly dilated esophagus may be mistaken for an enlarged heart or a mediastinal mass (see Fig. 2). When such an esophagus



(a) (b)

Fig. 2. (a) Flat chest plate suggesting enlarged heart. (b) Same patient with barium in dilated esophagus.

is filled with barium, the diagnosis is unmistakable. In no other condition is the dilatation of the esophagus so immense. The appearance of the lower end is characteristic. There is a round, smooth lower end which narrows to a point, through which from time to time a fine trickle of barium is seen passing into the stomach. If the level of fluid is raised in the esophagus or if the patient exhales completely, the barium will spurt into the stomach. When the fluid has returned to its former level, the cardia closes and shuts off the stream of barium. Taking a deep breath and holding it produces a similar effect. Retention of food particles in the lower end of the esophagus sometimes gives rise to irregular contours of the walls, which may be mistaken for carcinoma (see Fig. 3).

Some writers believe that esophagoscopy is unnecessary in the majority of these cases. Others advocate esopha-

goscopic examination in every case because of the possibility of overlooking a carcinoma or ulcer. This would seem to be the wiser and more logical course to follow, because both complications have been known to occur, and each would necessarily modify the treatment. We use esophagoscopy in every case.

TREATMENT.

Treatment is directed toward allaying inflammation of the esophagus and improving passage of food into the stomach.

Fig. 3. Huge achalasia of esophagus with food in lower end, producing shadow suggestive of carcinoma.

The patient initially is put on a diet of bland liquid and soft food. Highly seasoned foods or foods with coarse roughage such as seeds or skins are forbidden.

Very severe cases, with a marked esophagitis and a tendency to run a septic temperature, should be handled as advised by Canfield.[8] The esophagus is irrigated with 1,000 cc. of hot physiologic saline solution one to three times a day. A temporary gastrostomy may be done, and the patient fed through it, or he may subsist on intravenous fluids for a few days. Because the idea of spasm has been widely accepted for a long time, antispasmodics such as tincture of belladonna (15 drops three times daily after meals) are usually prescribed. There is growing criticism of this medication.

Since many of these patients are quite nervous, a mild sedative is definitely beneficial. Phenobarbital (½ gr. three times

daily) is usually given our patients. Unquestionably, phychogenic factors and nervous strain play an important part in many instances. One patient dated the onset of her trouble from the death of her father. Another progressed satisfactorily until he had some financial difficulties, which caused a marked aggravation of his symptoms. Still another patient was a tense, nervous type with a history of trouble associated with considerable nervous strain for 10 years. After treatment for a year her general condition had improved sufficiently to have an adenoma of the thyroid removed. Her basal metabolic rate was +3. Since then she has had no trouble, eats anything and has had no further dilations. This patient did not receive thiamin chloride. Some writers view these psychogenic factors as being of primary importance.[15,60] On the other hand, achalasia of the cardia has been reported in the newborn.[1,3,50] When psychogenic elements are present, they should receive every consideration and should be controlled insofar as it is possible.

Since Willis fashioned the first crude dilator, the most important part of the treatment has been dilation of the cardia. Many semiflexible and flexible nonexpanding dilators have been designed since that time.[25,59] A number of expanding dilators with a rubber balloon which can be filled with water or air have also been used.[6,28,45,49] At present the two best dilators are the mercury-filled, flexible dilators of Hurst[25] and the mercury-filled, pneumatic bag of Browne and McHardy.[6] The latter is preferable for most cases because it can be passed as easily as any other dilator and permits greater dilation than the nonexpanding type. The intervals between dilations depend on the response to the treatment. At first we give one dilation a week, and later they are given only when symptoms recur.

TECHNIQUE.

For the comfort of the patient and to facilitate dilation, a sedative and atropine should be given before the dilator is passed. The throat should be partially anesthetized by a 4 per cent cocaine spray. Three sprays from an atomizer at intervals of a few minutes usually suffice. The index finger guides the bag into the hypopharynx with the patient in the sitting position, with the neck extended and the chin forward

and up. As soon as the bag passes the cricopharyngeus muscle area, the neck is bent forward and downward, so that any saliva or regurgitated material will gravitate into an emesis basin held by the patient. This prevents material from entering the larynx and causing a fit of coughing. The dilator then usually passes of its own weight down the esophagus and into the stomach. In most cases there is a momentary hesitation at the cardia before the bag passes into the stomach. In a few patients definite resistance may occur to the passage of the dilator through the cardia. To overcome the resistance one must use a slight steady pressure. Because of the danger of rupturing the esophagus no real force should be used even with flexible dilators unless the dilation is done under the fluoroscope.

The fluoroscope also should be used when there is some doubt that the dilator is actually passing through the cardia. In cases with marked dilatation and sacculation the dilator may kink in the esophagus and not enter the stomach. This is readily apparent under the fluoroscope. After it has entered the stomach, the bag is inflated to a pressure of 6 pounds and gently pulled back until it impinges against the cardia. The air is then released and the dilator pulled back about 6 cm. so that the bag is in the cardia proper. It is then inflated to 12 pounds, or less if the patient experiences pain, and left in position for several minutes. The bag is then deflated and the dilator is withdrawn. Force should never be used either in pushing the dilator into place or in withdrawing it because fatalities may result from either procedure.

Observations on the use of dilators vary greatly. Walton[58] stated that in all of his cases resistance occurred at the cardia, both to the passage of the dilator into the stomach and to its withdrawal. Because of this, he favored the theory of spasm. The opposite experience was reported by Hurst,[23] who stated that in the majority of his patients resistance was not encountered; therefore, he favored the theory of achalasia. Our series included both types. In the majority of cases in which resistance was encountered pain was a prominent symptom. This pain occurred both spontaneously and upon manipulation.

The question naturally arises whether or not spasm actually exists in these instances. Another possibility is that

fibrosis at the cardia interferes with its distensibility. One of our patients undoubtedly belonged in this category. She was 63 years old, gave a history of eight years' duration and had an enormously dilated esophagus. She was so emaciated and had such a severe secondary anemia that she was admitted to the hospital for treatment. After one or two unsuccessful attempts at dilation, she was placed under the fluoroscope. The direction of the tip of the dilator was watched and a firm, steady pressure maintained until the dilator finally passed into the stomach. As the cardia gave way she experienced a very severe pain, which almost made her faint. After this she showed rapid progress, and subsequent dilations were performed easily. She weighed 97 pounds when she was first admitted and gained 39 pounds in five months. The last interval between dilations was one year.

VITAMIN B_1 AS AN ADJUNCT IN THERAPY.

Because of the logical way in which Etzel's theory of vitamin B_1 deficiency fitted all the known factors in cardiospasm or achalasia and because of the brilliant results reported by Stinson in his four cases, thiamin chloride was used on a series of cases to evaluate its efficacy. The possibility suggested itself that if nerve degeneration were due to thiamin chloride deficiency, some measure of normal function might be restored if treatment were instituted before degeneration had progressed to an irreversible stage. This would depend on the duration and severity of the condition. Accordingly, about 2½ years ago thiamin chloride was added to our regular schedule in the treatment of these cases. The following is a comparison of the results obtained in this group with the results in a similar group treated prior to the use of thiamin chloride.

Initially, the thiamin chloride is injected intramuscularly in doses of 3,000 to 6,000 I.U. (10-20 mg.). The injections are given every second or third day in conjunction with the other recognized procedures such as diet and dilations. When the condition improves so that food passes with relative ease into the stomach without retention and stagnation in the esophagus, the thiamin chloride is given by mouth in doses of 10 mg. three times a day, 15 minutes before meals. It is

important that the thiamin chloride be given intramuscularly as long as there is retention of food in the esophagus, because if given by mouth during this phase it will not be absorbed properly and benefit will be minimal.

RESULTS.

Questionnaires were sent to 13 patients who had not received thiamin chloride, seven of whom failed to reply; 25 questionnaires were sent to patients who did receive thiamin chloride, with no response from six. The number of patients in the two groups was disproportionate because *1.* the first group (no thiamin chloride) was composed of patients who did not follow through with treatments and were not sufficiently interested to answer the questionnaires; and *2.* those who did follow through with their treatments were subsequently put on thiamin chloride. Many of the second group are still under observation.

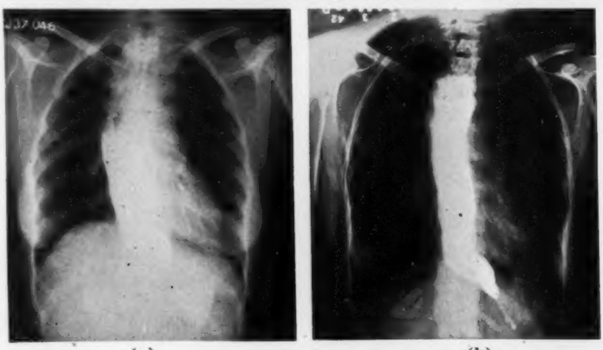
Of the six in the nonthiamin chloride group, two had excellent results, three had good results and one had fair results. They had received from two to 22 dilations, with an average of 11. Of the 19 in the thiamin chloride group, 11 had excellent results, six good and two fair. Of the two fair results, one did not continue her treatment very long and received dilations at home with a small dilator. The other was far advanced and returned for only two dilations. These patients received from one to 24 dilations, with an average of nine. Both of these groups are too small to permit statistical studies or hard and fast conclusions. The evaluation of the results depends entirely upon the patient's own estimate and, therefore, is subject to a certain amount of error.

One patient with good results had a 10-year history and extreme sacculation at the lower end of the esophagus. He had a vitamin B peripheral neuritis with marked weakness in his legs. He had been under treatment for 18 months before receiving thiamin chloride in large doses but had been given vitamin B complex and liver, and the neuritis had cleared up. He felt that the injections of thiamin chloride actually relaxed his spasm.

Another patient with a 16-year history, admitted by ambulance in February, 1942, weighed 93 pounds. She weighed

139½ pounds in July, 1943, and progressive Roentgenograms showed a definite diminution in the size of the esophageal dilatation (see Fig. 4).

A young woman, with a history of four years' duration, showed a reduction in the size of her dilatation to practically normal after treatment for about eight months (see Fig. 5). There were also primary peristaltic waves actually progressing to the cardia. These waves were not present at the time of her first examination. She had had the condition only four years; and since the dilatation was moderate, the disease



(a) (b)

Fig. 4. (a) Degree of dilatation of esophagus at beginning of treatment; 16-year history. (b) Same case; 17 months later.

was not far advanced. She is not symptom-free as yet, however, and still returns occasionally for dilation. This may represent the versible reaction for which we have been seeking. One patient who has had excellent results showed no demonstrable reduction in the size of the dilated esophagus after 7½ months. Progressive Roentgenologic studies are being continued on these patients after treatment for a sufficient length of time.

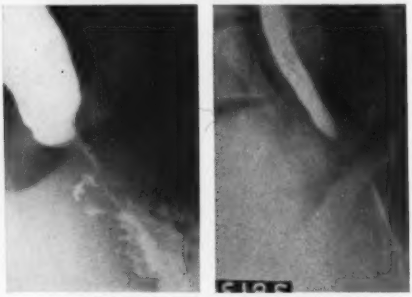
From the results in the relatively small group of cases studied it is apparent that these patients are much better generally than those who did not receive thiamin chloride. The use of this substance, however, does not displace the use of the other measures which have been beneficial.

CONCLUSIONS.

A brief survey of achalasia of the cardia is presented, together with a small series of cases treated in the orthodox

manner with the addition of thiamin chloride to their regimen. It is believed that thiamin chloride is an important addition to the treatment of achalasia of the cardia, but that it cannot replace all other forms of treatment.

It may be of great significance that two patients have shown definite diminution in the size of the esophagus. In one of these patients the lumen has returned to approximately normal size and showed a return of the primary peristaltic wave. One far advanced case with an excellent symptomatic



(a) (b)

Fig. 5. (a) Lower end of esophagus and cardia showing typical achalasia in a patient with a history of four years' duration. (b) Same patient eight months after beginning of treatment, showing marked reduction in size of esophagus.

result showed no change in the size of his esophageal dilatation.

The possibility is suggested that this condition may be reversible if treatment can be instituted early enough before the degeneration of the nerve mechanism has progressed too far. The well-being of the further advanced cases can be improved.

This is a preliminary report. Further study is required to establish the true value of this addition to the treatment of achalasia of the cardia.

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TRAUMATIC DEAFNESS; PROBLEMS OF PREVENTION.*

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In the process of evolution, when animal life began to leave the water, its primitive semicircular canals with their function of controlling balance and freedom of movement became more highly specialized. During this period of development and adaptation to a new environment, the gill clefts gradually disappeared, and a process somewhat resembling a fourth semicircular canal evolved; as it grew in length, it curled on itself and assumed a snail-like shape. No true organ of hearing was present in this transitional stage. As animals became more terrestrial and less amphibian, there began to develop sensory organs which could perceive sound waves. Through these evolutionary changes, after aeons of time, the organ of equilibrium evolved into the true organ of hearing. This was the last one of the sense organs to develop.

It is doubtful whether any organ of the human body has so little power to recuperate from injury, either chemical or mechanical, as has the sense organ of hearing. It is of interest to note that the VIIIth nerve, subjected as it is to overstimulation by sound, is in addition more frequently injured by foreign substances circulating in the blood than any other of the nerves of special sense. Nager[1] stated that the enchondral layer of the otic capsule remains in its embryonic phase of development throughout life, that the vital processes of bone consisting of perpetual resorption and deposition are absent, and that vascularization is almost absent. He added that all these factors are evidence of a greatly reduced vitality because of the unalterable nature of the enchondral layer.

Until the invention of gunpowder, the harnessing of steam, and the utilization of power by means of compressed air and

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electricity, probably the loudest noise to which the human ear was subjected was the clap of thunder or the roar of some cataclysmic convulsion of nature such as a volcanic explosion. The human ear was never intended to experience the noises associated with warfare and some of the occupations of the present day. Biology teaches that every species of animal life is adapted to its environment; all of its organs are fitted to its functions and all of its functions to its environment. Man has no means of insulation against acoustic trauma; he has nothing that is homologous to the musculature or valves that aquatic animals have for keeping water out of the external auditory canal. The basal turn of the cochlea, which is the location of sound perception for the high notes, is in close juxtaposition to the oval window; consequently it is a vulnerable part of the acoustic mechanism, for, as Dickson and Ewing[2] noted, it is exposed to the initial violence of thrusts of the stapes in response to impulsive sound.

Probably there is no subject pertaining to human physiology, anatomy and preventive medicine that offers such a challenge today as does the protection of this immediate area against acoustic insult. Since Fosbroke,[3] in 1831, presented his paper on traumatic deafness, monographs on this subject have been legion. As early as 1918, Guild[4] in an article entitled "The Otopathies of War; with Especial Reference to the Traumas Due to Air Waves," mentioned in the bibliography 177 articles. Bunch,[5] in 1937, in a paper entitled "The Diagnosis of Occupational or Traumatic Deafness; a Historical and Audiometric Study," cited 160 articles. With the additional scientific research since that time, there has been a virtual renaissance in this subject. All phases of it have been developed, and especially has exhaustive study been made of acoustic trauma incident to occupational deafness and war deafness as well as to that resulting from the general noises of civilian life in keeping with the increased tempo of the times.

Since the introduction of audiometry, it has become possible to measure sound by a more accurate method than ever before. The rustle of leaves, nature's voice in the quiet garden, the peal of thunder, and the damaging noises of the pneumatic drill, the riveting machine and the airplane engine may now be accurately determined in relation to the thresh-

old of hearing. By means of the audiometer it has been established that sound becomes painful at from 115 to 130 dcb.,[6] depending upon the frequency. The intensity level of some of the frequent noises that cause acoustic trauma is: riveting or chipping hammer, 115 to 140 dcb.; airplane motor (1,600 revolutions per minute, 18 feet from the propeller), 115 dcb.; airplane propeller, 120 dcb., and a stream of air under high compression with a 5-inch diameter outlet, 125 dcb. or more.[6,7]

The literature is replete with reports such as the one made by Barr,[8] in 1886, of a survey of the hearing of 100 boilermakers who had followed their occupation over a period of three or more years. In no single instance was normal hearing present; in 90 per cent of these subjects the reaction to the Rinne test was positive. Jobson[9] noted the presence of some degree of deafness in 100 per cent of 60 subjects, chosen at random, who had served at the front in the first World War, but had been away from it long enough to have recovered from any temporary effects upon the hearing. The estimated 40,000 aural casualties of that war will be greatly exceeded by those of the present war, in which at least six times as many members of the American armed forces are participating. The present estimate is that 250,000 cases may be expected.[10]

In an exceedingly large percentage of persons returning to civilian life with defective hearing, the impairment will undoubtedly be the result of traumatic deafness caused by gunfire, the noise of Diesel and airplane engines, and the numerous other hearing hazards incident to warfare of today. An evidence of the large number of deafened casualties expected is the following statement in a communication of the War Department, issued Feb. 18, 1944:

"Hearing aids will be furnished by the Medical Department to the personnel in active military service who are suffering from hearing defects that preclude the performance of military duty, regardless of line of duty status, when examination shows that such aids will materially improve the hearing of the individuals concerned."

In this communication it is further stated that the prescribing and fitting of hearing aids will be limited to the

Deshon General Hospital, Butler Pa., the Borden General Hospital, Chickasha, Okla., Hoff General Hospital, Santa Barbara. Cal., and to other hospitals for the rehabilitation of the deaf which subsequently may be designated. To these hospitals will be referred at the earliest practicable date those patients with stationary or progressive deafness free from acute inflammatory aural disease whose loss of hearing acuity in the better ear is 30 dcb. within the conversational range. Again, in a large percentage of these cases the deafness will be due to trauma.

There are few subjects in medicine in which the etiology and pathology are better understood than in traumatic deafness. The deafness is the result of degeneration of the hair cells of the organ of Corti; with injury, they degenerate and become absorbed, and once these cells degenerate, the loss is permanent. As Lurie[11] stated, the degeneration is not confined to the particular part of the organ of Corti originally damaged but spreads and may continue for long periods of time after the original injury.

In 1866, Chippendale[12] recommended the wearing of cotton in the ears for persons encountering gunfire and added that this precaution was used in France "by artillerists to prevent deafness and bleeding of the ears." The United States is now in the third year of the second World War, and still cotton, which is known as the least efficient of all ear defenders and which gives an insulation of from 7 to 12 dcb., is the insulator that is generally used and accepted. I have interrogated scores of naval officers, and invariably their replies have been that they are advised to put cotton in their ears, stand on toes and keep their mouths open until all guns have completed firing. The following is a letter from an officer of the Navy, who is under my observation for traumatic deafness:

"May 19, 1944.

"I have looked high and low and in between for definitive information concerning ear plugs, care and protection for the ears, and there seems to be no information or preventive protection except what I found in a circular letter issued by the Bureau of Medicine to the flight surgeons.

"Subject: Prevention of Auditory Traumatism and Resultant Loss of Hearing.

"*Gunfire:* Ear defenders may be worn if available. Otherwise carefully applied, close-fitting cotton plugs should be worn and not removed until all guns are known to have completed firing. (Ear plugs or defenders can be obtained from the Mine Safety Appliances Co., Pittsburgh, Pa.).

"Wholesale inquiries to persons of authority produced nothing but negatives concerning the subject. However, it seems to be an unwritten law in the case of aviators that they should use cotton. This at their own discretion and preference. . . .

"I would like to be of more service to you in regard to this matter, but I am sure if there is any common knowledge about the subject, aside from the meager details I have given you, I certainly would have found it. There just isn't any."

One cannot but wonder whether or not the otologist has assumed his responsibility in this important branch of preventive medicine. Has he in his research and laboratory work been as alert as has the immunologist in the prevention and, indeed, almost the elimination of rabies, smallpox, diphtheria, typhoid fever and other diseases that blight mankind?

There may be some explanation for the seeming lack of interest and required co-operation in the study of acoustic insulation, but the consideration of some of the phases of the subject may hurt. Hundreds of thousands of dollars have been expended in the study of the amplification of sound and in laboratory research directed toward the development of and improvement in hearing aids. This phase has had its mercenary attractions, and the results of the efforts put forth represent today a most remunerative field.

The otologist has stimulated the interest of the physicist, and great scientific organizations such as the Western Electric and Westinghouse companies have pioneered in this field. The fact that the sales value of the modern hearing aid approximates $100 while that of the ear defender varies from a few cents to a dollar or two is a pecuniary aspect of this subject not to be disregarded. It may have had some influ-

ence which might make it appear, unpleasant as it is to admit, that someone, otologist or militarist, or vice versa, might have stimulated the other to make something more effective in the field of prevention.

One deterring factor may have been that the attainment of a state of perfection in an acoustic insulator for use in every type of environment, occupation and combat is perhaps impossible. Inability to accomplish the impossible has until recently made the scientist, whether otologist, militarist or physicist, content to let the matter remain in its unacceptable, inefficient state. The soldier in the jungle of New Guinea searching out the sniper camouflaged among the fronds of a coconut palm could hardly by wearing an acoustic insulator so handicap his alertness that he could not hear the rustle of a leaf or the snapping of a twig. Neither could one expect an ear defender to shut out a noise such as was heard in Australia and in England, each more than 2,500 miles distant, when the great eruption of 1883 blew away the volcanic island of Krakatao. Within the wide range between such extremes there is, however, a place for the acoustic insulator. Co-operative research and study will eventually perfect this needed means of protection for the aural mechanism.

The statement of Brunner[13] that as long as war lasts, traumatic deafness will exist and that the only way to eliminate it is to do away with war, is true; so will there always be tetanus, smallpox and diphtheria. That vast progress has been made in immunization against such diseases admits of no argument today. Like progress in the avoidance of traumatic deafness can be made if there is hearty co-operation between militarist, otologist and physicist in the vast sound laboratory which is the world of today. A propitious moment for such investigation is at hand. The price of one torpedo or pension paid one deafened war casualty would provide the necessary funds to defray the expense of a complete study that would at least advance the science of acoustic insulation to a state of perfection equivalent to that of the modern hearing aid.

It seems that the present emergency has stimulated an interest in the subject of the prevention of war deafness, which has been more or less latent since the close of the last

war. My attention has been directed to a Navy communication dated Oct. 1, 1943:

"The insulation value of cotton varies from 3 to 18 dcb. for all frequencies, whereas the maximum corresponding protection afforded by ear defenders ranges from 30 to 40 dcb.

"It has been demonstrated that long-continued exposure to the noise of Diesel engines leads to a permanent loss of auditory acuity. In addition to such actual loss, the noise and confinement have a deleterious effect on the nervous system and general well-being of the man, increasing nervous tension and fatigue. These conditions are reproduced in certain types of aircraft. Also exposure to the high intensity, high frequency 'cracks' of the anti-aircraft and double-purpose guns by their crews often results in auditory traumatism.

"Ear defenders are being procured and are to be distributed to the crews of the anti-aircraft and double-purpose guns and to the Engineer Forces of Diesel-propelled ships. One hundred per cent replacement will be allowed.

"During the period that the ear defenders are being placed in production, the Mine Safety Appliances Ear Defenders may be procured."

The Carnegie Corporation, in October, 1925, granted a subsidy of $90,000 for the study of otosclerosis. If this Society could develop such enthusiasm for the prevention of traumatic deafness as was manifested at a dinner given by the late Dr. A. B. Duel, in 1924, on the occasion of starting an endowment for the study of otosclerosis, this subject would not be in the undeveloped stage it now is. Many preventive measures could doubtless be devised in the construction of various war machines which would lessen the acoustic trauma to which aviators and submarine personnel in particular are subjected.

During this wartime emergency, as a result of my close contact with the personnel of a number of military camps, shipyards and air bases, I have seen a large group suffering from traumatic deafness and have had the opportunity of studying some of these sources of traumatic deafness. I believe that if every member of this Society would come in immediate contact with riveters and operators of chipping

hammers even for a brief period of time, or would spend some time on the firing line of some of the gunnery schools where the 50-calibre machine gun is being used, they would all reach the same conclusion that I have reached, that cotton as an ear defender is as obsolete and out of date as is the stagecoach. Compare the insulation of cotton with that afforded by such defenders as the one developed by Knudsen, the S. M. R., and the plastic model advocated by Lieut. Cox and Lieut. Geller, and cotton as an ear defender would be condemned as virtually useless.

At the Naval Air Gunnery School in Jacksonville it became necessary to rotate the officers who served as instructors on the firing range because of damage to the ears. These men, who were on the firing range for eight hours daily, complained so much of tinnitus, earache and deafness that another type of ear defender was deemed necessary because cotton had proved most inffective. Lieut. Cox and Lieut. Geller, of the Dental Corps, developed a plastic mold made of a hydrocolloid material. The use of the plastic defenders silenced complaints, and they supplanted cotton with good results. It is recognized there, and is true also of other schools, that many of the men suffer from traumatic deafness even before they leave the gunnery school.

The statement often repeated in the literature that workers in the shipyards and various factories will not use ear defenders has not been borne out in my experience with them. Persons who have been furnished ear defenders have refused to work without them and have recommended their use to associates. I believe that those who will not wear ear defenders are persons who are already suffering from a high degree of nerve deafness and who experience neither pain nor inconvenience from the loud noises of chipping hammer, riveting hammer and other machinery. Persons who are just starting in a noisy occupation and are suffering from pain, tinnitus and headaches, and those in the premonitory stages of a nerve deafness welcome ear defenders.

On the gunnery range, my experience in firing 50-caliber machine guns led me to conclude that the plastic plugs of Cox and Geller and the defenders developed by Knudsen reduced the intensity of the sound to the point where the

noise was encountered without discomfort. I was also surprised at the ease with which one could converse with the gunner. Both at the gunnery school and at the shipyard where I was in close proximity to the chipping hammer, cotton packed in the ears afforded virtually no protection against painful noise.

The audiogram shown in Fig. 1 illustrates what may occur during a few weeks' course at a gunnery school. In this case a nerve deafness had developed before the patient had com-

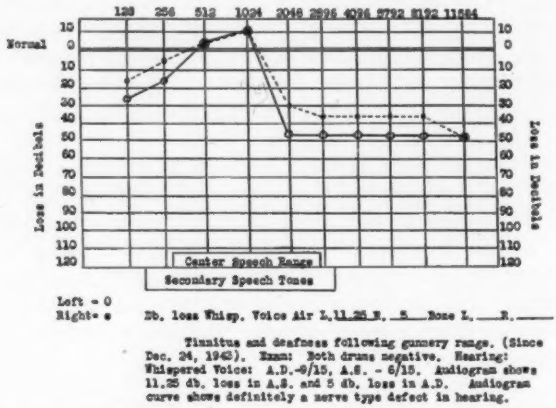

Fig. 1. Audiogram of a soldier suffering from tinnitus and deafness following gunnery practice. Examination of both drums gave negative results. Whispered voice: A. D., 9/15; A. S., 6/15. The audiogram shows a loss of 5 dcb. in the right ear and 11.25 dcb. in the left ear. The audiogram curve indicates definitely a nerve type of defect in hearing.

pleted his gunnery training and while he was still far from an active theater of war. The condition might have been avoided had proper acoustic insulation been provided.

Ten days ago it was my privilege to visit one of the aviation fields in Florida. One of the medical officers informed me that 75 per cent of the deafened aviators who had come under his personal observation were those who are stationed in the Sperry upper local or top gun turret of the Flying Fortress. An inspection of this turret provided an explanation, for it is well known that loud noises in an enclosed chamber are much more conducive to damage. In the course of our survey of some of the acoustic problems encountered in the Fortresses, a pilot grounded because of deafness was

questioned. He had 2,000 flying hours to his credit and had accumulated 500 hours of combat flying within 11 months. He became aware of difficulty in hearing after 400 flying hours. The audiogram shown in Fig. 2 offers evidence of the type and degree of deafness.

I am officially advised in a letter dated May 20, 1944, that the Army at this time is not recommending any particular type of ear plug. Dr. Walter A. Wells, of Washington, D. C.,

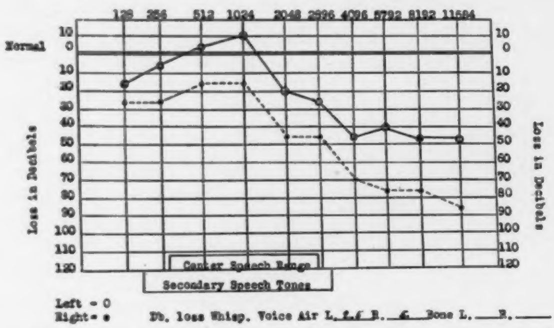

Fig. 2. Audiogram of an aviator grounded because of deafness. Credited with 2,000 flying hours, and with 500 combat hours in a period of 11 months, he had normal hearing before flying, but began to be aware of difficulty in hearing after 400 hours of flying.

wrote to me, May 2, 1944, that the Navy now recommends the defender of soft synthetic rubber developed by Knudsen, which is manufactured by the Mine Safety Appliances Company, of Pittsburgh, and which will soon be in mass production. This information seems not to have been widely disseminated. Cotton plugs and the plastic defender developed by Cox and Geller are the only ones now in use at the Jacksonville Naval Air Base.

A full description of the Knudsen defender is given in an article entitled "Ear Defenders," by Norman A. Watson and Vern O. Knudsen,[14] of the University of California at Los Angeles, which was published in the *Journal of the Acoustical Society of America* in January, 1944. In a personal communication I received from Dr. Knudsen a few days ago, he expressed the opinion that this defender would provide as much protection against impulsive blasts as against continu-

ous sounds, for, he added, a reduction of 30 dcb. should certainly reduce most explosive blasts to a harmless level.

Whether the best ear defender has yet been made can only be determined by further study. Certainly here is a field for fruitful research. A perfect ear defender must meet many requirements. It must be easy to insert and have good retention; it must be comparatively light in weight and unbreakable; it must decrease noise of high frequency without impairing to an important degree the hearing of commands. Cleanliness must be especially considered because of the mycotic infections common to the tropics. Shea[15] recommended the use of oleum ricini as a protection against otomycosis.

SUMMARY.

An effort is made to present some of the problems of traumatic deafness and to stimulate progress in prevention. No discussion of the pathology is necessary before this Society. The necessity of adequate acoustic insulation for members of the armed forces and for workers exposed to the noises of industry is emphasized. It is noted that in this third year of the second World War interest in this phase of preventive medicine is being manifested, and progress in prevention is beginning to be made. Cooperation between the otologist, the militarist and the physicist in making a constructive approach to this pressing problem is of paramount importance.

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